THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—35TH YEAR. SYDNEY, SATURDAY, JULY 17, 1948. No. 3.

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INTRACRANIAL DISORDERS OF THE NEW-BORN ASSOCIATED WITH BIRTH.[1]

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THE subject of the neonatal intracranial disorders related to birth has always attracted attention, not only because of the immediate risk to life which is entailed, but also because of the possible sequel of cerebral damage. The prevention—when possible—and treatment of these conditions impose heavy responsibility upon both obstetricians and pædiatricians.

These intracranial disorders may for convenience be classified under three headings: (i) intracranial hæmorrhage; (ii) anoxæmia (asphyxia—apnœa—hypoxæmia); (iii) fracture of the skull.

THE INCIDENCE.

The figures by Agnes Macgregor of Edinburgh[(1)] (Table I) indicate the incidence of the various causes of death (including still-births) in a series of 1071 autopsies on infants in the neonatal period. The statistics of most large centres are in agreement with these. Intracranial hæmorrhage caused 27% of deaths, anoxæmia (asphyxia) 24% of deaths, infection 20% of deaths, congenital defects 20% of deaths and miscellaneous conditions 9% of deaths. It will be seen that intracranial hæmorrhage and anoxæmia caused 51% of the deaths.

CHANGES IN THE INTRACRANIAL CONTENTS DURING BIRTH.

As the skull passes down the birth canal, it is subjected to (*a*) compression and (*b*) elongation. The contents of the skull are the brain, the cerebro-spinal fluid, the blood vessels, the circulating blood, and the fibrous septa—the *falx cerebri* and the *tentorium cerebelli*—with their enclosed venous sinuses. The blood and cerebro-spinal fluid are readily displaced, and the soft brain is easily moulded. The falx and tentorium are the unyielding structures, and if stretched beyond the limits of their elasticity they will tear. The falx is perforated anteriorly in a cribriform manner, so that there is more "give" in this structure than in the more rigid *tentorium cerebelli*. At the posterior attachment of the falx to the upper surface of the tentorium, the fibres decussate to either side of the mid-

TABLE I.

Nature of Death.	Nature of Birth.		Total.
	Premature.	Full Time.	
Stillbirth	235	218	453
Neonatal deaths	436	182	618
Total	671	400	1071

line. The triangular area so formed is the weakest part of the tentorium, and it is estimated by McGuinness[(2)] that this area is injured in over 92% of deaths from intracranial hæmorrhage.

If the head is compressed in the antero-posterior diameter, there will be a consequent increase in the height of the skull with stretching upward of the tentorium and the possibility of its tearing. If the head is compressed in the lateral diameter, there will be a consequent increase in the antero-posterior diameter with stretching of the falx. This would also be transmitted to its attachment to the tentorium, again with the possibility of a torn tentorium.

The unsupported veins, as they pass from the brain to the sinuses, may also be torn, so that the great vein of

[1] Read at a meeting of the Obstetric Society of the Women's Hospital, Melbourne.

Galen as it passes to the straight sinus, and the cerebral veins as they pass to the superior sagittal sinus, may be ruptured. Thus the following structures in the skull are the most likely to be damaged by the hazards of birth: (*a*) the *tentorium cerebelli*, (*b*) the unsupported veins and (*c*) the *falx cerebri*.

Prolonged severe pressure on the head, after rupture of the membranes, will squeeze blood from the cranial contents and produce varying degrees of anoxæmia of the brain.

It is to be remembered that in premature infants the walls of the veins are immaturely developed and are lacking in both muscle and elastic tissue. These veins therefore rupture more easily than do the veins of full-time infants.

In post-mature infants, the skull bones are more ossified than normal and therefore the skull is less easily moulded than is that of the normal infant.

While intracranial hæmorrhage and anoxæmia are discussed as separate conditions, clinically it is found that the two conditions frequently occur together. In a given case it may be difficult to assess what symptoms are due to the hæmorrhage and what are due to the anoxæmia. Of the two conditions the anoxæmia is much the more serious, not only as the greater cause of death, but also as regards subsequent cerebral spoiling.

Intrauterine anoxæmia is frequently revealed alone at the post-mortem examination; but it is sometimes difficult in a case of intracranial hæmorrhage at autopsy to decide how much associated anoxæmia has also been present.

Intracranial Hæmorrhage.

Intracranial hæmorrhage in the new-born is venous in character, and may be of the following four types: (i) subdural, (ii) extradural, (iii) subarachnoid, (iv) intraventricular. Of these types only the subdural and extradural are really traumatic, the subarachnoid type being associated with anoxæmia, and the intraventricular type with anoxæmia and toxæmia. The last-mentioned two types are mostly found in premature infants.

Subdural Hæmorrhage.

Subdural hæmorrhage is the commonest type. In Craig's series[3] this variety accounted for about half the cases of intracranial hæmorrhage. Tears in the tentorium were present in 66% of his cases of intradural hæmorrhage, other sources of hæmorrhage being the great vein of Galen or the cerebral veins, or less frequently the transverse sinus, the straight sinus and the superior and inferior sagittal sinuses.

The tentorium frequently tears at the free edge, the hæmorrhage becoming infratentorial and passing down around the brain-stem and medulla. Should the tear be lateral, the hæmorrhage tends to be supratentorial and to pass upwards over the occipital and temporal lobes. Sometimes a hæmatoma may occur between the two layers of the tentorium, but this is not important clinically.

Subdural hæmorrhage, if of any considerable extent, breaks through the arachnoid into the subarachnoid space.

Infratentorial hæmorrhage is more serious than supratentorial hæmorrhage, since the pressure of the effused blood about the vital centres in the medulla may prove fatal. Infratentorial hæmorrhage tends to occur with tears in the free edge of the tentorium, and with rupture of the great vein of Galen and the inferior sagittal sinus. At times the hæmorrhage is both infratentorial and supratentorial.

Causes of Subdural Hæmorrhage.

The causes of subdural hæmorrhage may be divided into (i) ante-partum, (ii) intra-partum and (iii) post-partum causes.

Ante-partum causes include injury to the *fœtus in utero* as by external version and possibly blows to the abdomen.

The intra-partum causes are as follows. Long, difficult, spontaneous labours or difficult instrumental deliveries are sometimes to blame. In breech birth, the rapid change in shape of the skull allows insufficient time for moulding, or undue suprapubic pressure on the skull during birth may be responsible. Precipitate labour also exposes the infant to the risk of insufficient moulding of the head. Prematurity entails a double risk of intracranial hæmorrhage, owing firstly to the immature state of the walls of the veins and secondly to the low blood prothrombin level of the premature infant. The practice of "holding up the birth" by holding the head back till the arrival of the obstetrician constitutes a real and unnecessary risk. Finally the cord, if wound tightly round the neck, may cause considerable venous congestion with risk of hæmorrhage.

The post-partum causes of intracranial hæmorrhage include such conditions as fracture of the skull sustained subsequent to birth, and hæmorrhagic disease of the new-born.

The Symptoms and Treatment at Birth.

Should the hæmorrhage be very extensive, still-birth may result. If it is less severe, the infant is born in a state of asphyxia, either *asphyxia pallida* or *asphyxia livida*. In this state of apnœa, the anoxæmic state of the respiratory centre prevents it from functioning. It has been shown that while a physiological degree of anoxæmia will stimulate the respiratory centre to function, a pathological degree renders it functionless. Since the state of apnœa is highly dangerous to the baby, it is important that methods of resuscitation should be carefully considered.

In the care of the infant with *asphyxia pallida* there are four indications to be met. (i) Since the infant is in a state of shock, he must be kept warm. (ii) The upper air passages must be cleared. (iii) Oxygen must be supplied. (iv) Chemical stimulants must be given if necessary.

The warmth can be supplied by warm blankets, or if it is preferred, by warm baths.

It is desirable that the clearing of the upper air passages should be done aseptically. The ordinary method of "sucking out" the mucus with a catheter, from which the mucus is subsequently blown out by the operator's breath, is unhygienic. Reintroduction of the soiled catheter exposes the infant to the risk of infection and subsequent broncho-pneumonia. The bulb of a rubber ear-syringe or breast pump *et cetera* can with advantage be substituted for the operator's mouth as a source of suction. When it is available, a suitable electric pump is a great boon. (It is particularly necessary in the treatment of all babies delivered by Cæsarean section that the liquor and mucus should be removed from the infant's upper air passages. In normal deliveries, the pressure of the birth canal on the chest, after the delivery of the head, squeezes out liquor and mucus, but the infant born by Cæsarean section misses this. It is therefore recommended that these babies should be inverted and that the trachea should be "milked" in order to rid the baby of any fluid in the respiratory passages.)

The supplying of oxygen is an urgent matter. The gas can be given by a variety of methods of varying efficiency. Oxygen diffuses very rapidly, so that as long as it is absorbed into the blood, the particular route used is of no great moment. Oxygen may therefore be absorbed from the bronchial, pharyngeal or gastric mucosa, and to a slight extent from the skin. Intratracheal insufflation is beyond the technical skill of most practitioners. Furthermore, it would appear from physiological data that pressures which are capable of inflating the alveoli are dangerous and may cause rupture of the alveoli and terminal bronchioles, with the production of interstitial emphysema or pneumothorax, while lesser and safer pressures are inadequate. Also the principle involved is unphysiological, in that oxygen is being forced in with a positive pressure before the thoracic cage has expanded, whereas in normal conditions the air is sucked in, owing to the negative pressure produced by the expansion of the chest.

The simplest manner of supplying oxygen is by means of an intranasal catheter. The rate usually advised is about six bubbles per second. The other nostril and the mouth are kept closed.

Face masks can be used, either operating at a constant pressure or fitted with a mechanism for interrupting the flow.

Respirators of the "E and J" type with face masks are found to be satisfactory in use. They work at a positive pressure of 17 centimetres of water and a negative pressure of 11 centimetres.

Respirators of the "Drinker" type are also used. Respirators of these types are usually within the financial scope only of the larger institutions.

The old method of mouth-to-mouth insufflation, the air in the cheeks being used as in blowing out a match—the usual pressure being five to eight centimetres of water—is often efficient; but the risk of infection to the infant is very real.

It is proved that the carbon dioxide content of the blood of asphyxiated infants is above normal, so that "Carbogen" is contraindicated in resuscitative measures on the infant who is born apnœic. Later, when respiration has been established, it can be advantageously employed to increase the respiratory amplitude.

Since, in the apnœic infant, the respiratory centre is unable to function, those chemical stimulants which act on the chemo-receptors—the carotid and aortic bodies—are indicated. "Lobeline" is the most effective of these. The weak strength, containing three milligrammes per millilitre, is used. One millilitre is given to a full-time infant and 0·5 to 0·25 millilitre to a premature infant. (Since this drug is rapidly excreted—within half an hour—its administration can be repeated as required.) It is most advantageously injected into the umbilical vein and then "milked" down the cord into the infant's circulation.

Nikethamide ("Anacardone", "Coramine") is a less effective respiratory stimulant, and is less readily excreted, probably taking more than an hour to be eliminated; 0·5 millilitre is usually given to a full-time infant and 0·25 millilitre to a premature baby. This drug overcomes the action of the barbiturates, and this action may be helpful if the mother has been heavily sedated with barbiturates. Symptoms of overdosage are not infrequently encountered. The infant is restless and "strung-up". It sneezes and has a peculiar barking cough. Twitching may be present.

It is emphasized that artificial respiration in an infant who has never breathed is quite useless. Since the lungs are unexpanded, there can be no squeezing out of air and elastic recoil. Such measures also expose the infant to unnecessary trauma and chilling.

The infant with *asphyxia livida* requires the same treatment as for the pallid asphyxiate, in addition to cutaneous stimulation. This may be supplied by the application of cold to the skin or by the old-fashioned spanking.

Symptoms after Birth.

Later the infant with subdural hæmorrhage may present a variety of symptoms. Frequently he is restless and sleepless, showing a high degree of agitated activity and distress. The shrill high-pitched scream, "the cerebral cry", is probably indicative of intense headache. Frequently there is a moaning type of respiration, and wild darting movements of the eyes are often seen.

Other infants present a different picture. They are somnolent, apathetic or stuporose, with an associated pallor and flaccidity. Such symptoms tend to occur with supratentorial hæmorrhage.

Head retraction and opisthotonus frequently occur in association with infratentorial hæmorrhage.

Ocular symptoms, such as nystagmus, unequal pupils, ptosis or strabismus, are sometimes observed.

Rigidity of one or more limbs, with the thumb held tightly in the palm and the fingers flexed, may be encountered. (One often meets new-born babies who hold the hands with the wrists flexed and the fingers extended in the position assumed by the "choreic hand". This is not due to cerebral trauma, but appears to be a developmental condition. It is observed most often in premature babies and disappears spontaneously.)

Paresis of a limb or of one side of the body may be seen. Twitching and convulsions are frequently encountered, and clonic movements may occasionally occur.

Because of the interference with the respiratory centre, symptoms of atelectasis are frequently noted—cyanosis, which may be continuous or intermittent, with irregular shallow respiration and chest retraction.

Vomiting, often of a projectile character, is a common symptom. At times the baby may vomit large amounts of thick gastric mucus. He may be unable to suck and in severe cases may also be unable to swallow.

Sweating is often seen, and frequently there is a rise in temperature.

Craig[4] described an "adder tongue" in these infants—that is, a constant protrusion of the tongue. Although this has been looked for, I have not observed it. This may be due to the fact that the routine administration of adequate fluids which these infants now receive has prevented symptoms of dehydration such as this adder tongue from occurring.

If the intracranial hæmorrhage is of any great extent, there is a palpable increase in tension of the fontanelle.

Complications.

The complications which may occur are atelectasis, bronchopneumonia, dehydration fever and urinary infections.

Atelectasis is often the immediate cause of death.

It is not uncommon for infants who have required much resuscitation to develop bronchopneumonia in the first four or five days of life. Mouth-to-mouth insufflation is often responsible for this, as is also the infection caused by frequent "sucking out" by a mucus catheter over a period of hours. Intranasal catheters are frequently not sterilized. They are often left hanging on the side of the oxygen cylinder and are not always innocent of contamination with the floor. Similarly, used mucus catheters are left lying about on the tables or the bed clothes. One feels that much of this lung infection could be prevented if all catheters used for the respiratory system received the same aseptic care as the infant's feeding utensils, and some mechanical means of sucking out mucus could be employed.

Dehydration fever may be observed in those infants who vomit or are unable to suck or swallow, and who have not been given adequate fluid.

Urinary infection likewise is generally associated with dehydration and may be prevented by administration of sufficient fluid.

Diagnosis.

The diagnosis is made on the history, the symptoms, the condition of the fontanelle, and the results of lumbar puncture. Useful information is given by the feel of the fontanelle. The most helpful method is to select a normal baby from the nursery for comparison, and with this control baby and the patient both lying at the same angle in their cots, to palpate the fontanelles of both simultaneously. In this way mild differences in tension can be appreciated. In normal infants the fontanelle has a fluid feel. In moderately raised tension, the fontanelle feels like sponge-rubber. (In two such cases the pressure of the cerebro-spinal fluid has been found to be 200 millimetres of water.) When there is a great increase in tension, the fontanelle feels hard and woody, and in very severe cases it may bulge. One must be cautious in interpreting the tension of the fontanelle if the tissues above it are thickened and œdematous, as occurs when the caput is in this area.

Lumbar puncture is a most useful diagnostic aid and is the only means of distinction between intracranial hæmorrhage and conditions producing similar symptoms due to a great increase in the amount of cerebro-spinal fluid, without hæmorrhage.

In normal new-born infants the pressure of cerebro-spinal fluid is low—namely, 14 to 80 millimetres of water—and the amount of fluid is small, so that it is often difficult to obtain cerebro-spinal fluid. The greater the increase in tension of the fluid, the easier is the lumbar puncture of performance. If the puncture is done with the infant sitting up, the procedure is simplified, since the spinal theca is more distended. The correct position of the infant is essential for the successful puncture. The sitting position means much less disturbance for the baby than if

he is placed on his side and flexed, and his respiration is not so embarrassed. The baby is lifted on to a firm table with his legs straight out in front. He must be held quite straight and the weight of the unsupported head will help to flex the spine. The needle is inserted in the usual position, and so long as it is straight in the mid-line no difficulty will be experienced in entering the theca, which is usually reached at a distance of about three-quarters to one inch. One does not usually feel the snap of the needle entering as one does in older patients.

If there is considerable tension and the blood-stained fluid is not too thick, it may even spurt from the needle. If there is no great tension, or if the fluid is thick with blood, one may need to wait up to a quarter of a minute before the fluid emerges. It is always wise to have ready a sterilized syringe which can be attached to the needle, as the fluid may be so thick as to require aspiration. It is wise also to use a fairly stout needle—gauge 20 or more.

If after one has waited for fifteen to twenty seconds and has attempted aspiration no fluid is found, the needle is rotated, and if there is still no fluid the needle is introduced further. The commonest error is to wait too short a time for the emergence of the fluid, and to push the needle in too far, so that it strikes the plexus of veins on the anterior aspect of the spinal column, and a "bloody tap" results.

Once fluid is obtained the infant is laid down on his side with the head level, and the flexion of the spine is stopped.

The amount obtained will vary with the tension and the viscosity of the blood-stained fluid. The fluid may be thick and dark, or it may be bright red, or only slightly blood-stained. In cases in which the hæmorrhage is older, it may be xanthochromic (occasionally the cerebro-spinal fluid in normal infants may be yellow particularly in jaundiced infants or premature babies).

In my experience the normal cerebro-spinal fluid of the new-born baby is almost always crystal clear. Roberts,[5] in his series of 423 punctures on Negro babies performed in the first twenty-four hours of life, found the fluid coloured yellow in every case.

It may be necessary to distinguish between blood in the cerebro-spinal fluid originating from an intracranial hæmorrhage, and the blood from the trauma of the lumbar puncture. The main differences are set out below.

Lumbar Puncture Trauma.	*Intracranial Hæmorrhage.*
The fluid clears as it flows.	The fluid remains uniformly blood stained.
On standing or centrifugation the supernatant fluid is clear.	If the hæmorrhage is over 12 hours old the supernatant fluid is yellow.
The benzidine test applied to the supernatant fluid gives a negative result.	The supernatant fluid gives a positive response to the benzidine test.
Microscopic examination shows: (*a*) no crenated red blood cells or ghost cells;	Microscopic examination shows: (*a*) crenated red cells and ghost cells if the hæmorrhage is over twelve hours old.
(*b*) no excess of white cells.	(*b*) excess white cells.

Generally the clearing of the fluid as it flows and the microscopic examination of the fluid are the most useful clinical signs.

The old idea that the lumbar puncture involved so much handling as to distress the child is not correct. With practice and the employment of the sitting position the handling can be reduced to a minimum and the baby is not adversely affected.

Treatment.

In any case of difficult delivery it is wise to give the mother vitamin *K* analogue during labour. The infant should also be given vitamin *K* injections at birth and during the first four days of life. The raising of the blood prothrombin above the lower level which occurs physiologically in the first four days in full-time infants and in the first ten days in premature infants, will diminish the tendency to hæmorrhage.

Since it has been shown that the effect of vitamin *K* lasts for forty-eight to seventy-two hours, it would seem ample to give the injections at intervals of forty-eight or at most twenty-four hours. In premature babies, in whom the hypoprothrombinæmia may last till the tenth day, vitamin *K* can with advantage be given at birth and again on the third and sixth days.

Lumbar puncture, besides being a diagnostic measure, is also a therapeutic procedure. If the tension is greatly raised the relief of tension is life-saving. It is sometimes objected that the lowering of the increased tension will start hæmorrhage again, but clinically this is not found to be so. Theoretically also this objection is not borne out, since it is known[6] that raised intracranial tension causes a local rise in pressure in the cerebral vessels and this will increase hæmorrhage.

It is sometimes difficult to know how much fluid to remove. If a manometer is available and the infant is horizontal and quiescent, thus allowing an accurate reading, it is probably safe to lower the tension to about 100 millimetres of water. If a manometer is not available, one is guided by the tension of the fontanelle and by the rate of drip of the cerebro-spinal fluid. Roughly speaking, one stops when the fluid drops at the rate of one drop every three to four seconds.

It is usually recommended that the lumbar puncture should be repeated daily till the fluid clears. The object is to remove as much blood as possible, the blood being regarded as an irritant. However, in cases in which the tension of the cerebro-spinal fluid drops to normal after the first puncture, or in which the fluid is thick and viscous with blood, it is often difficult to obtain even very small quantities of fluid, so that the amount of blood removed is negligible. In such cases repeated puncture is not employed.

The position in which the infant is nursed is of importance. The head of the cot is raised, since this will lower the pressure in the venous system of the cranium.

The baby should be handled as little and as gently as possible. He should be fed, changed and sponged in his bassinet, and should not be treated as an ordinary baby till four days or more after all active symptoms have disappeared.

Should there be any suggestion of atelectasis, or should cyanosis be present, oxygen is a prime necessity. Not only will the anoxæmia be prejudicial, but the vasodilatation associated with cyanosis will increase the hæmorrhage.

Sedatives will be required to combat the restlessness and twitching or convulsions when present. Adequate doses are necessary, and the average six to seven pound infant is given 0·25 grain of phenobarbital, which can be repeated in four hours or as need arises. I have found phenobarbital the best sedative.

The administration of fluid and food requires attention. Those infants who can suck and swallow present no difficulty. The infants who cannot suck, but who can swallow, are fed from a spoon. Those infants who cannot swallow may be fed by gavage. If they will not tolerate the gavage tube, or should vomiting be troublesome, fluid must be given parenterally. While this may be given intravenously, the continuous subcutaneous or intramuscular drip is safer and easier. The outer aspects of the thighs are the most convenient sites, and fairly coarse hypodermic needles are used. Because of the functional inefficiency of the new-born baby's kidney and the difficulty it experiences in concentrating electrolytes, it is unwise to use normal saline solution. I employ one-fifth normal saline solution and 4% glucose. Contrary to the earlier teaching, the glucose is not irritating locally. The usual rate is half as many minim drops per minute as the baby weighs in pounds for the first day. This gives one and a half ounces per pound of body weight per day. If this route is still required after two or three days or so, the rate can be increased. If nothing is being taken by mouth the rate is increased to as many minim drops per minute as the baby weighs in pounds (three ounces of fluid per

pound of body weight per day). A Y-piece with a needle in both thighs is used, and the fluid can be clipped off from time to time if necessary. As was mentioned earlier, adequate fluid is necessary to prevent dehydration.

If there is obvious atelectasis, or if the infant has required much resuscitation, penicillin may be given for the first two or three days as a prophylactic measure. The usual dose is 5000 units every three hours for a full-time baby, and 3000 units every three hours for a premature baby.

The question of subdural aspiration and intraventricular puncture may arise. If there is indubitable evidence of increased intracranial tension, and if lumbar puncture reveals fluid under normal tension, subdural aspiration is indicated. The head is shaved and the infant is placed flat on his back with the head securely held. It is wise to have a stop on the needle one centimetre from the tip. This is introduced to a depth of one centimetre parallel with the horizontal plane through the coronal suture at a point well lateral to the lateral angle of the fontanelle. This is done on both sides. In the normal infant only a few drops of cerebro-spinal fluid escape—more than one cubic centimetre is said to be abnormal. When there is considerable hæmorrhage the blood-stained fluid may escape under considerable tension. It is not wise to remove more than about 15 cubic centimetres at one time.

If greatly increased intracranial tension is obviously present, and if both lumbar puncture and subdural aspiration reveal no great increase in cerebro-spinal fluid, a ventricular puncture is indicated; but in these cases, as will be mentioned later, the prognosis is poor.

The role of surgery in cases of intracranial hæmorrhage is always a matter of controversy. Since the majority of subjects have a torn tentorium, the hæmorrhage is in a position inaccessible to the surgeon, and the repeated lumbar puncture—"the lumbar decompression" of some writers—usually serves. Clinically, it is found that in the majority of cases of moderate intracranial hæmorrhage the symptoms have usually cleared by the fifth or sixth day, and no localizing signs of nervous involvement are detectable, nor are signs of increased intracranial tension. Surgical treatment is not considered for these infants.

If after six days or so the symptoms have not disappeared, the position must be reviewed. We should endeavour to determine whether the persisting symptoms are due to the hæmorrhage or to the effects of an associated cerebral anoxæmia. If there are localizing signs such as weakness or rigidity of a limb or of a side of the body, or if subdural aspiration has disclosed the presence of much blood-stained fluid, the symptoms are probably due to the hæmorrhage and the question or surgery should be considered. It is usually agreed that new-born infants stand craniotomy badly, and this is therefore not advised. It is probably sufficient at this stage to make a trephine opening on the appropriate side (or sides) and to wash out blood and clot with saline solution. The subsequent management is outside the scope of this paper.

Sequelæ.

In following these patients subsequently, one is struck by the absence of sequelæ in a large proportion. There is no doubt that the risk of hæmorrhage to a child's subsequent cerebral development is much less than the risks involved in anoxæmia. Roberts[5] followed a series of patients suffering from proved intracranial hæmorrhage with symptoms at birth and found 66% of them to be normal. The possible sequelæ in intracranial hæmorrhage are well known—spasticity, paresis, mental defects, epilepsy, chorea, athetosis, hydrocephalus *et cetera*. As has previously been mentioned, it is often difficult to assess how much damage is due to the hæmorrhage and how much to the existing anoxæmia.

Ingraham and Matson[7] have directed attention to the damage caused by subdural hæmorrhage over the cerebral vertex. A false membrane may form over the adjacent cerebrum preventing it from growing, or the effused blood may absorb fluid into it so that the encysted collection becomes continually larger, pressing on and distorting the brain.

Extradural Hæmorrhage.

Extradural hæmorrhage is rare, and may occur in cases of fracture of the skull bones, or in rupture of a sinus. In the former case the treatment is the treatment of the fracture. Cases of ruptured sinus are usually fatal.

Subarachnoid Hæmorrhage.

Subarachnoid hæmorrhage is anoxæmic and non-traumatic in origin. It occurs mainly in anoxæmic premature infants with atelectasis, and a predisposing factor is the immature state of the walls of the veins. Vascular oozing occurs, particularly over the vertex. There is an excessive amount of cerebro-spinal fluid, and the brain appears to be encased in a reddish-yellow jelly-like layer. Blood in this area is readily absorbed. It gives rise to no definite symptoms and is not by itself a cause of death.

Intraventricular Hæmorrhage.

Intraventricular hæmorrhage is always a serious condition. Though it is occasionally encountered in full-time babies, it tends to occur mainly in premature infants who are anoxæmic and atelectatic, and whose mothers have suffered from toxæmia—either toxæmia of pregnancy or intercurrent toxæmia from infection *et cetera*. The source of hæmorrhage is usually a vein in the lateral ventricle, though it may sometimes come from the chorioid plexus.

The symptoms most commonly come on during the first day, but may occur up to three weeks. A vivid description is given by Craig.[2] The onset is sudden and dramatic with great distress. The child utters a piercing cry suggesting agonizing headache. The colour becomes grey and the respiration panting. Rolling of the eyes and nystagmus occur. At times the eyes protude. The infant lies with the trunk rigid, the hands clenched and the legs drawn up. Twitching and convulsions occur. The fontanelle is tense and bulging. Vomiting and a high temperature are usually present. The condition is fatal within two or three days or less, and at post-mortem examination the hæmorrhage may be found to have ploughed its way right through the substance of the brain.

Treatment consists of the administration of vitamin *K*, ventricular puncture, and the administration of oxygen.

Anoxæmia (Asphyxia, Apnœa, Hypoxæmia).

Oxygen lack is the most serious hazard to which the infant is subjected during the process of birth. He is equipped better than older individuals to withstand poor conditions of oxygenation and it has been shown that new-born infants who have not breathed for ten to fifteen minutes can survive. The adult does not survive seven to eight minutes of apnœa. The resistance of the new-born infant is due to its low cerebral metabolism, to its anaerobic source of energy (from anaerobic breakdown of glycogen) and to its poikilothermia.

However, if the oxygen supply to the tissues falls below the minimum level, serious damage will result. It is well established that of all the tissues in the body, the nervous system is the most sensitive to oxygen lack. It has been further shown that different levels of the brain exhibit differing degrees of sensitivity to anoxæmia, the cortical neurones being the most susceptible at the time of birth. It is possible, therefore, for the infant to be born apparently healthy, with the respiratory centre functioning well and with no obvious signs at birth, and yet to have gross spoiling of areas of his cerebral cortex, a condition described by Schrieber[6] as *"asphyxia occulta"*. On microscopic examination these "devastation areas" show as pale areas with spoiled or degenerated ganglion cells.

In Macgregor's series anoxæmia caused 24% of all deaths, and it was significant that it was three times as common among the still-born babies as among those who died in the neonatal period, which shows its lethal effects.

It is unfortunate that the term "asphyxia" is used, since it carries with it to most minds the implication of suffocation or of respiratory obstruction, when actually the mechanism is different.

Mechanism of Anoxæmia.

The accepted mechanism of anoxæmia is as follows. The oxygen lack stimulates the vasomotor centre, causing a rise in blood pressure. This increases the resistance to the work of the left side of the heart, so that ultimately the left side fails. There is therefore an accumulation of blood in the pulmonary circulation, with failure, dilatation and engorgement of the right side of the heart and the venous system. The rise in carbon dioxide content also causes venous dilatation.

Anoxæmia also affects the capillary walls directly, causing first a loss of tone with congestion and then a spoiling of the wall so that serum exudes through; later the further spoiling allows a diapedesis of red cells from the capillary.

Pathology.

The pathological findings are thus easily understood—the multiple subserous petechial hæmorrhages, the extreme venous congestion with dark fluid blood, the dilated right side of the heart and the œdematous congested organs. The type of brain described by Craig[8] in his series as "hæmorrhage into the brain substance", in which congestion and multiple small hæmorrhages occur, probably falls in this group. The bronchi often contain inhaled meconium.

The Causes.

Cerebral anoxæmia may be caused by a variety of conditions, which can be divided into (*a*) local causes in the skull, (*b*) factors producing a general fœtal anoxæmia, in which the brain shares.

Local pressure on the head may be produced by any of the following: (i) long, difficult, spontaneous delivery after rupture of the membranes; (ii) difficult instrumental delivery; (iii) the practice of "holding up the birth" with pressure on the head; (iv) direct prolonged suprapubic pressure on the skull during breech birth.

The following factors may be responsible for the production of general anoxæmia, in which the brain is involved.

1. Any interruption of the placental circulation—for example, premature separation of the placenta, retroplacental hæmorrhage, knots or pressure on the cord, prolonged and frequent contractions—may produce general anoxæmia.

2. Post-maturity may be another factor. If the pregnancy continues for more than two weeks over term there is risk of trouble, depending on the rate of the degenerative changes taking place in the senile placenta. It was the custom of the late Arthur Wilson of Melbourne to divide post-mature pregnancies into the following two types. The first is that of the mother, generally aged under thirty-five years, whose placenta continues to function well, and whose baby continues to grow in size; in this type the danger lies in an over-large baby with disproportion. The second type is that of the mother, often aged over thirty-five years, whose abdomen begins to shrink in size and who loses weight, owing to absorption of liquor and wasting of the fœtus. It is in this second type that the fœtus runs a grave risk of cerebral damage. It is known that the post-mature or "senile" placenta becomes infarcted—that is, clotting takes place in those intervillous spaces from which the syncytial layer disappears, thus cutting off some of the fœtal blood supply. Also there is endarteritis of the vessels of the villi which produces a similar result. Thus the fœtus is subjected to an ever-increasing cutting off of oxygen (and other essentials). A stage is reached when the more vulnerable cortical cells in the brain undergo irreversible changes, and the infant who is born at this stage will grow up with some degree of cortical atrophy.

Should the condition persist *in utero* past this stage the fœtus will ultimately die.

The infant who is the result of a post-mature and senile placenta is easily recognized. He stands the experience of birth badly owing to his harder, less easily mouldable skull and to his preexisting anoxæmia. He is usually long and thin, with a long thin neck. His finger-nails and toe-nails are long. His skin is free of vernix, dry, inelastic, leathery and peeling, often in large flakes. The new pink, thin, skin of the finger-tips and toes often protudes like fingers from old worn-out gloves. The liver is usually smaller than normal. These babies are lethargic and behave rather like premature infants as regards taking their food. They frequently exhibit gross signs of cerebral irritation with increased amounts of cerebro-spinal fluid. Some die in the first week or so in an athreptic state.

3. The third factor is premature senility of the placenta. In this condition the placenta begins to degenerate before the appointed time, so that in a full-time or even in a premature infant similar changes to those met with in the post-mature infant (with the exception of the long nails) are found. Since the changes are really those of intrauterine malnutrition, it is obvious that they can be met with at any stage of gestation. If the placentæ of these infants are examined, they are usually found to be small, leathery and generally infarcted.

4. Maternal toxæmia is another factor. Faber[9] has recently drawn attention to the possible association of maternal toxæmia with later cerebral atrophy. The infarction of the placenta and the retroplacental hæmorrhages are held responsible. When one considers the large number of cases of toxæmia and the very small number of cases of cerebral damage resulting from them, one concludes that this risk is fortunately not great.

5. Massive maternal blood loss must affect the amount of oxygen available to the fœtus, and exposes it to grave risks of intrauterine anoxæmia. (If in addition the villi are torn, the fœtus itself will lose blood from its own circulation).

6. The intrauterine brain damage associated with fœtal erythroblastosis will not be considered here.

7. Analgesics and anæsthetics used in labour may be involved. These may affect the fœtus in two ways. Firstly, anything which depresses the mother's respiratory centre, producing maternal apnœa, may affect the fœtus, causing the "*asphyxia occulta*" of Schrieber. Secondly, these substances may act on the infant's own respiratory centre, lowering its sensitivity and producing apnœa.

Irving, Berman and Nelson[10] found that when no drugs were used the percentage of apnœic infants was 2%. When sedatives were used, the figure immediately jumped up to 35% (Table II).

TABLE II.

Sedation.	Percentage of Apnœic Infants.
No drugs	2
Pentobarbital ("Nembutal") and ether (given rectally)	35
Pentobarbital ("Nembutal") and scopolamine	37
"Sodium Amytal" and scopolamine	39
"Sodium Amytal" and ether (given rectally)	41
Pentobarbital ("Nembutal") and paraldehyde	50

Eastman has found that with nitrous oxide and oxygen anæsthesia, concentrations of oxygen less than 15% are dangerous to the fœtus and should not be used. Continuous caudal analgesia is outside the scope of the ordinary practitioner and carries with it a high rate of forceps deliveries. The perfect sedative or analgesic is yet to be found.

In all cases in which the mother is given sedatives or analgesics, not only should the fœtal heart be watched, but the depth and frequency of the mother's respirations should also be observed. Should the respirations become shallow or infrequent, she should be given oxygen. One has only to watch the improvement in colour which takes place in the infant delivered with a pulsating cord, when the mother is given oxygen, to appreciate the therapeutic efficiency of this measure. Similarly in any case in which the mother is having prolonged contractions in a long labour, the giving of oxygen to the mother is a safeguard for the baby. The time to treat intrauterine "asphyxia" is during labour. By the time the baby is born the damage is done.

Symptoms of Cerebral Anoxæmia.

If cerebral anoxæmia is severe, the infant is still-born. If it is less severe, the infant may be born in a state of *asphyxia pallida* or *asphyxia livida*. Infants who have suffered from cerebral anoxæmia frequently show cerebral symptoms, usually within the first four days of birth. This condition is variously designated "cerebral œdema", "cerebral irritation", "acute external hydrocephalus". It is is probably due to injury to the capillary walls by the anoxæmia, so that fluid passes through, causing œdema of the brain and its coverings and an increase in the amount of cerebro-spinal fluid. It is not uncommonly seen in infants delivered by Cæsarean section, often an elective operation, and is apparently due to the apnœa encountered in these babies due to the maternal anæsthesia.

In infants who die of this condition there is found an excessive quantity of clear cerebro-spinal fluid with œdematous brain and meninges, so that the vessels appear to be floating in liquid gelatin.

The symptoms of this cerebral œdema are clinically indistinguishable from those of intracranial hæmorrhage, except that there is an absence of localizing signs which may be present in hæmorrhage. The infants are usually agitated, restless and distressed, with a cerebral cry. The respirations are often shallow and irregular and chest retraction is seen. Darting movements of the eyes may occur, and head retraction is generally present. Twitching and convulsions are common symptoms and there is an increased tension of the fontanelle. If it is unrelieved, the condition often proves fatal.

The diagnosis is made by lumbar puncture, when crystal-clear cerebro-spinal fluid escapes usually under considerably increased tension. It may spurt out in a stream two to three inches from the needle.

Treatment.

The treatment consists in lumbar puncture and the administration of oxygen. The relief given by the lumbar puncture is most dramatic and frequently life-saving. It may need to be repeated in twelve to twenty-four hours, but a third puncture is rarely required.

The value of lumbar puncture in these cases both diagnostically and therapeutically is great. It is impossible to distinguish the condition from intracranial hæmorrhage by any other means, and an infant may be allowed to die under the mistaken idea that it is suffering from intracranial hæmorrhage with a hopeless prognosis unless a puncture is performed.

The possible sequelæ of severe cerebral anoxæmia are well known—the cerebral atrophies, cerebral palsies, mental defects, spasticity and a gross variety of neurological conditions. It has been suggested that a possible sequel of anoxæmia persisting after birth is failure of the *ductus arteriosus* to close. It has been shown that increased oxygenation in fœtal guinea-pigs caused the closure of the ductus by muscular contraction, whereas diminution of oxygen caused opening of the ductus. Suggestive experiments have also been performed on infants on this point. The milder grades of cerebral anoxæmia have a correspondingly better prognosis with regard to escape from cerebral complications.

Fracture of the Skull.

Linear fractures are generally the result of difficult instrumental deliveries. It is the associated conditions of anoxæmia and hæmorrhage which dominate the picture and call for treatment.

Depressed fractures of the skull—the pond or spoon-shaped fractures—are usually the result of the fœtal head's being driven by the contracting uterus against the prominent sacrum or symphysis. A depressed fracture should be elevated, and the sooner this is done after birth, the easier it is. Pressure with the fingers on the surrounding bone may be successful, or it may require more skilful surgical manipulation.

Conclusions.

If we take it that 50% of intracranial hæmorrhages (the subdural and extradural) are traumatic and 50% (the subarachnoid and intraventricular) anoxæmic, Macgregor's figures can be given as follows: traumatic intracranial hæmorrhage, 13·5% of deaths; anoxæmia, 37·5% of deaths. This emphasizes the importance of oxygen lack to the fœtus as a cause of death. The figures are just as striking as a cause of cerebral symptoms in later life. Schrieber, in 500 cases in which cerebral symptoms were present, found a history of apnœa in 70% of the cases in which the infant's condition was known at birth.

It is by efforts directed to the cutting down of fœtal anoxæmia, and to the prevention and early adequate treatment of intracranial hæmorrhage, that we can make our biggest contribution towards reducing the number of cases of neurological damage.

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AN EPIDEMIC OF GASTRO-ENTERITIS IN INFANTS, WITH SPECIAL REFERENCE TO TREATMENT.

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GASTRO-ENTERITIS (summer diarrhœa) is an endemic disease of infancy in Brisbane. It is generally mild in nature and responds readily to simple dietetic and, when indicated, intravenous hydration therapy. During the period from 1936 to 1941 an average of 78 infants were admitted to the Brisbane Children's Hospital each year suffering from this complaint. The mortality rate during this period was 5·6%. During the war years, concomitantly with a vast increase in city population and resultant overcrowding of the city area, the number of admissions to hospital increased. The average annual admissions during the period from July, 1941, to July, 1947, was 254 and the mortality rate was 5·2%. It is interesting to note that hospital records reveal a similar rise in the number of admissions during the latter part of the First World War and in the early post-war period, the mortality rate then being as high as 31% (1918-1921, average 312 admissions *per annum*).

During the year 1947, 384 infants (children aged under two years) were admitted to this hospital suffering from "gastro-enteritis". This number represents only those infants who were ill enough to require admission to hospital, or whose home circumstances or facilities were considered inadequate for home care. During the year many infants of necessity have been

treated from the out-patient department alone. It is intended to limit the present discussion to a consideration of patients admitted to hospital only.

In November and December, 1946, and in the early months of 1947, despite the usual routine therapy, we noticed a gradual but increasing rise in the mortality rate of this disease. Many children admitted to hospital were more ill than those to whom we were accustomed. They responded readily to hydration and dietary therapy, but many of them were subject to frequent and severe relapses. Post-mortem examinations revealed acute inflammatory lesions of the gut in a large percentage of cases, indicating an increased virulence in the causative organism. Attempted cultures of pathogenic organisms from the stools gave repeatedly negative results, although this may have been to some extent the result of failure to use specific culture media during the early months. In approximately 40% of infants coming to post-mortem examination during these early stages was found evidence of varying degrees of infection in the mastoid air cells. Fluid described as "mucopus" became a common sight at post-mortem examination of one or both of the mastoid cavities, while in approximately 25% of cases "frank pus" was found. Culture of the pus revealed in almost all cases a mixed infection of which the streptococcus was the prevalent organism. Frequently staphylococci and pneumococci were also demonstrated. From one patient, who underwent mastoidectomy, was obtained a pure culture of pneumococci from swabs taken during the operation.

Thus in the early months of this year, besides the usual clinical picture of gastro-enteritis, we encountered acutely ill infants, at first responding to therapy but frequently relapsing. White cell counts, examination of the tympanic membrane and myringotomy usually provided no indication of mastoid involvement, nor was there any local evidence of infection. The pulse and temperature charts were also found to be of no use in estimating the condition of the mastoid cavities.

Thus the clinical picture alone had to be relied upon in the making of a provisional diagnosis of a mastoid infection in association with the infection of the alimentary canal. As this condition is considered a complication of gastro-enteritis (probably owing to vomiting in the recumbent position), the treatment instituted will be dealt with later.

During the month of May it became evident that the phase of mastoid involvement was dying out, but we were being presented with a new clinical condition. Very toxæmic, dehydrated infants were being admitted to hospital, who showed no great response to therapy. These infants passed more or less characteristic orange-yellow fluid fæces containing no blood and very little mucus. On microscopic examination the relatively small number of pus cells was the most notable feature. From these stools organisms of the Salmonella series were grown on culture. In all early cases the organisms were found to belong to group C and were identified as *Salmonella bovis morbificans*. More recently a few organisms of groups B and F have been cultivated, but by far the most toxic and dangerous organism encountered has belonged to group C.

Infants suffering from this infection were extremely "toxic". The usual methods of hydration and dietary therapy hitherto employed were found to be inadequate, and despite repeated infusions of whole blood, serum, glucose, saline mixtures and Ringer's solution in association with rigid dietary measures, the mortality rate for this infection was recorded at 40%.

It is not our purpose here to discuss the signs and symptoms of gastro-enteritis. However, it is well recognized that in the early stages these may be somewhat indefinite and masked. This was especially so with the Salmonella infection, and as a result many infants suffering from this complaint were initially admitted to medical or surgical wards, where they remained until the true nature of their illness became apparent. This led to a degree of cross-infection in the hospital wards which had to be met by energetic and strict nursing precautions, disinfection *et cetera*. The high susceptibility of infants to the Salmonella organism was evident in ward cross-infection. It is also worthy of note that the vast majority of Salmonella infections, especially in the early stages, came from the more congested city areas, especially amongst families living grouped together in former military establishments.

In an endeavour to control spread of the infection, all patients yielding a Salmonella in culture material have been strictly isolated in hospital until three consecutive negative results have been obtained after the infant has made an apparent complete clinical recovery. Suffice it to say that these measures have during recent months greatly controlled the spread of the infection and have been a major factor in lessening the incidence of the disease. So far cultures of organisms of the Salmonella series have been obtained from the stools of 186 infants. Of these cultures 150 have belonged to group C.

The incubation period of *Salmonella bovis morbificans* infection appears to be from two to ten days. This figure has been arrived at from "trace-backs" to contacts of as many patients as possible. New-born babies have yielded positive stool cultures forty-eight hours after delivery, and these infants were presumably infected at birth.

Adults and children aged over two years seldom showed clinical evidence of any consequence when infected with this organism, and it is thought that in these groups the invasion is of a transitory nature. Of all patients treated 96% were infants aged under two years (Figure I).

A complete report on the epidemiology and bacteriology of this infection will be made later.

Up to the present 186 patients suffering from gastro-enteritis have given positive cultural findings for Salmonella infection. Reference to the graph (Figure II)

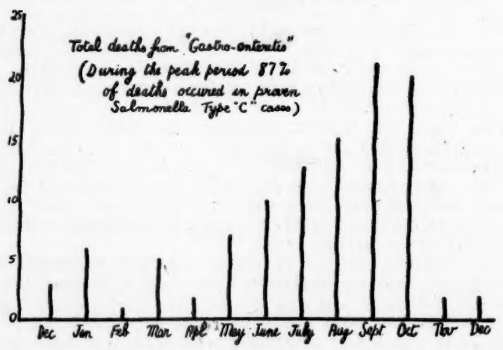

FIGURE I.

indicates the decline in the mortality rate from 40% to the present figure of 3% as additional methods of therapy have been employed. This dramatic fall may be due to some extent to an apparent decrease in the virulence of the infecting organism, as is evidenced by the decreasing percentage of very ill infants now being admitted to hospital.

TREATMENT.

The treatment of gastro-enteritis in infants may be discussed under the following headings: (i) maintenance of adequate hydration, (ii) maintenance of nutrition and protection against toxic liver damage, (iii) dietary measures, (iv) specific therapy for specific infections.

Maintenance of Hydration.

In recent years the maintenance of fluid and salt balance has become recognized as essential for normal cellular activity. It is estimated that loss of 10% of the water content of the body results in serious cellular dysfunction, while a loss of 20% results in death.

An eleven-pound baby weighs roughly 5000 grammes. Approximately 70% of this weight is made up of body fluids; that is, the fluid accounts for 3500 grammes. Thus

a loss of only 350 millilitres of fluid can result in serious cellular dysfunction. An infant suffering from anorexia with vomiting and diarrhœa soon loses this quantity of fluid, and the picture of dehydration (as evidenced by loss of skin elasticity and normal texture, sunken eyes and fontanelle, cerebral irritation *et cetera*) does not take long to develop. Thus hydration therapy in gastro-enteritis becomes an urgent problem, which must be undertaken at the earliest evidence of onset of the infection. In addition the body salts lost by the repeated vomiting and diarrhœa must be replaced immediately to assist in hydration and in the maintenance of normal cellular activity.

Owing to the technical difficulties of obtaining sufficient plasma from infants for repeated chloride estimations, little attempt has been made to carry out this procedure. Staff shortages and difficulties of supervision have prevented our making repeated collections of urine for estimation of urinary chloride content, and reliance has been placed on clinical observation as to the adequacy of salt replacement and as insurance against "over-salting" with resultant œdema. Both nursing and medical staff soon became conversant with the signs of dehydration or of over-replacement of previously lost salt.

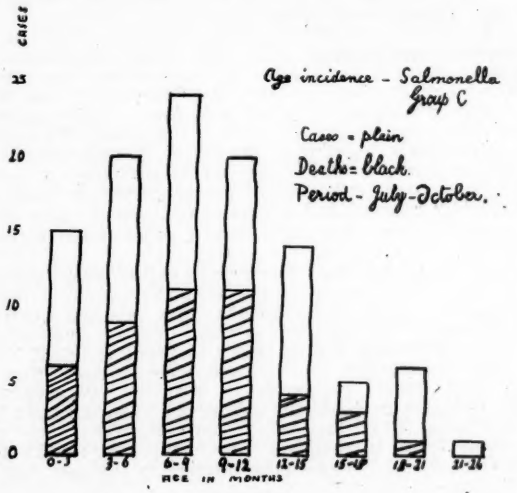

FIGURE II.

In health the normal infant requires up to two and a half ounces of fluid per pound of body weight *per diem*, to provide fluid for normal urinary output, skin and respiratory loss and normal stool loss. In the presence of fever, vomiting and diarrhœa this quantity is much increased, and in hydration therapy these factors must be taken into account. On the other hand, it is easy to "over-salt" and drown an infant while intravenous therapy is being undertaken. Constant supervision and adjustment of the rate of flow are essential when the intravenous route is being employed.

Method of Hydration.

The most reliable route for supplying fluid to infants refusing feeds or who have an inadequate intake is the intravenous route. The intramuscular and subcutaneous routes have been given a fair trial and have been found to be hopelessly inadequate, except for brief periods to assist a mildly dehydrated infant who is showing evidence of recovery by natural hydration, or in whom there is mild chloride loss producing what we believe to be a reflex anorexia.

Many of the infants treated received as many as ten to twelve infusions of fluid intravenously in a period of a few weeks. The chief disadvantage and most frequently encountered complication was found to be the development of infection with spreading thrombophlebitis. In the more debilitated patients this occurred rapidly. However, many infusions were kept running for as long as four to five days before evidence of infection became apparent. Despite the frequent occurrence of infective thrombophlebitis and the extreme debility of many of the infants, no post-mortem evidence of pulmonary embolism was encountered.

It is estimated that in health the average infant requires one to two grammes of sodium chloride daily. Daily caloric requirements were taken as 50 Calories per pound of body weight.

It has long been the custom at this hospital to combat dehydration and provide some of the caloric requirement of infants needing intravenous therapy by the routine use of half-strength physiological saline solution (usually called half-strength normal saline solution) and 5% glucose solution. To provide a higher caloric value and present more carbohydrate for liver function a higher concentration is desirable. However, higher concentrations predispose to infection and materially shorten the life of a "drip". In infants, whose veins suitable for intravenous therapy are few, "drips" must be maintained for as long as possible in each vein.

Early in the severe phase of the Salmonella infection fears of "over-salting" and resultant drowning of infants were entertained. As a result one-third normal and one-quarter normal saline solutions were given a trial, but were soon discarded as being entirely inadequate. With the use of half-normal solution grossly dehydrated infants could be adequately hydrated in a period of forty-eight hours; but when weaker solutions were used this result could not be obtained even with much longer periods of therapy.

The intravenous use of Ringer's solution (providing potassium and calcium ions) was found to provide no advantages and was discarded after a fair trial, especially when we were presented with two apparent cases of heart block.

Maintenance of Nutrition and Protection against Toxic Liver Damage.

All infants suffering from severe attacks of gastro-enteritis showed early evidence of gross falls in the serum protein levels, and it soon became evident that if these children were to survive every effort had to be made to maintain serum protein levels. This was especially the case with Salmonella infection, in which a low serum protein level was found to be indicative of extensive liver damage. At post-mortem examination the most striking feature in cases of severe toxæmia was the large, fatty, pale, degenerative liver in association with the usual inflammation of the gut. Microscopic examination of these livers revealed complete disorientation of liver structure with extreme fatty infiltration.

In the early stages of the Salmonella phase serum protein estimations were performed daily, and any patient found to have a fall in serum protein level was given a transfusion of serum or of whole blood if he was also anæmic.

In the majority of non-Salmonella infections these measures have been adequate, but in the toxic Salmonella infections it was found that large quantities of serum or blood would not elevate the serum protein level or prevent fatty degeneration of the liver. Massive and repeated doses of serum produced only a transitory rise in the serum protein level. A large series of toxæmic infants were given 100 millilitres of serum daily for periods up to six days without obvious benefit, even with an initial dose as high as 300 millilitres.

Thus a more adequate means of providing essential amino acids to protect and maintain liver function (and to assist in nutrition) had to be found. A commercially prepared casein hydrolysate ("Parenamine") was employed with most gratifying results. After a trial with four severely toxæmic infants, in whom it was assumed that fatty degeneration of the liver was inevitable, this substance was introduced intravenously to all infants requiring intravenous therapy. The normal infant requires three or four grammes of protein per kilogram of body weight per day. To all infants requiring intravenous therapy at least 15 grammes of protein in the form of amino acids were added to the intravenous "drip" daily. In addition, all

patients with low serum protein levels who were unable to utilize proteins given orally (presumably owing to a chronically inflamed gut) were given amino acids by the oral route. The effect was dramatic; provided liver change had not become irreversible, the serum protein level was not only maintained but was frequently elevated to normal. At about the same time more specific therapy (streptomycin) was introduced and the combination of these two measures reduced to 2% the mortality rate in uncomplicated Salmonella infection (that is, amongst otherwise normal infants displaying no other pathological abnormality). In the few subjects who have since come to post-mortem examination and who had received adequate therapy, the liver has been found to be macroscopically and microscopically normal.

Post-mortem examination of two infants infected with Salmonella of the group C series, whose death occurred only after a prolonged illness and who had received adequate amino acid therapy, revealed that the livers were normal. However, in both the gut was found to be chronically inflamed and apparently unable to digest and absorb normal nutritional requirements.

Finally, citrated whole blood transfusion has been employed whenever indicated. Many infants who had apparently recovered from the acute stages of the infection, but who failed to thrive, have progressed satisfactorily since the use of whole blood.

Dietary Measures.

The most important single factor in the treatment of gastro-enteritis is the feeding.

This is no easy problem, as the babies are usually listless, uninterested and often anorexic. In very ill babies the sight of a bottle will sometimes produce active resistance when otherwise all energy has been lost. Where this state of affairs is present recourse to intravenous therapy is indicated. From these severe cases all grades are encountered, and it is in the less severe grades that it is obvious that the first essential is a good nurse. These infants need to be coaxed to take their feeds, gently but firmly.

The child, whenever possible, must be nursed to be fed. He needs a sense of security and comfort, which is not given if he is fed while lying almost flat on his back in a position which he has probably occupied for hours. The best position for the child during feeding is to be lightly but firmly supported in the nurse's arms, comfortably propped, and sufficiently clad in clean, dry clothes to increase the feeling of well-being.

The type of teat used on the bottle is highly important. It must be firm enough not to "glue" together when it is sucked, and soft enough to make sucking as effortless as possible. Again, these babies have so little energy that if they are forced to work hard drawing milk through a minute hole in the teat they will soon give up the effort. On the other hand, if the hole in the teat is too large, the fluid will run unchecked down the infant's throat, and choking, swallowing difficulties, too rapid passage of the feed into the stomach and vomiting will occur.

The shape of the teat is important. If it is too long it reaches the back of the tongue and the pharynx and mechanical difficulties are obvious; if it is too short the baby cannot get a firm grip and exert adequate suction without undue effort.

It is important that all changes in diet must be very gradual; one cannot switch from whey to boiled skim milk in twenty-four or even forty-eight hours. There are numerous intervening steps which must be taken to achieve success, each step requiring anything from twenty-four hours to three or four days. This is the most trying and discouraging part in the treatment of these babies, and requires infinite patience on the part of the nursing and medical staff. Every step forward is taken cautiously and only when the preceding step has caused no adverse effect. One must be prepared to regress, progress and even regress again if necessary, the indication for regression being a return of vomiting, an exacerbation of diarrhœa or even a return of anorexia. This means, of course, that the child may be kept for a considerable time on a diet deficient in Calories and protein *et cetera*, and in these cases coexistent intravenous therapy is instituted, in the form of glucose and saline solution by the "drip" method, the administration of serum or of "Parenamine" or of both, and even transfusions of whole blood, for although there is no actual dehydration there may be starvation and hypoproteinæmia.

It is found frequently that an infant will take small quantities readily without vomiting and will apparently digest them, but if larger quantities are given, he refuses them or vomits. In these cases it is better to substitute two-hourly or even hourly feeds until the baby indicates his willingness to take more. This he will do when his gastro-intestinal tract is capable of dealing with greater quantities.

Nasal feeding is an occasional resort. It is particularly useful when anorexia is severe but vomiting and nausea are not features. In cases in which the two last-mentioned are evident, nasal feeding is usually a failure.

Rectal feeding of these infants has been found unsatisfactory, as has also the continuous intragastric drip method.

As for the actual diet *régime*, this must of necessity vary from individual to individual. Only broad lines of therapy can be indicated to act as a guide and cannot be rigidly adhered to in every case.

Breast-fed babies present little difficulty. They rarely contract gastro-enteritis and, when they do, usually all that is required is to take them from the breast, give them boiled or glucose water for twenty-four hours, then half-strength expressed breast milk for a further twenty-four hours, and then return them to the breast. Sometimes it is necessary to delay a day longer at each stage and even to insert a third stage of three-quarter strength expressed breast milk; but in general, breast feeding can be rapidly resumed and danger should then be past.

Artificially fed babies present a much more difficult problem.

Owing to the large number of patent foods on the market, there is a great variety in the nature and quantity of feeds to which any individual baby may have become accustomed. Unless there is some definite contraindication, it is usually deemed wiser to grade the child back to the type of diet to which he is accustomed. This is the feed which the mother is most used to preparing and to which no doubt she will resort when the child has been returned to her care. However, when there are obvious faults in the child's home diet, we usually find that the modified cow's milk mixture is the best feed to substitute.

The *régime* we have found to be most useful (after much trial of various *régimes*) is that involving the use of whey and whey-modified milks as indicated in Table I.

Whey is readily digested even by the sick premature baby and, though it is of little nutritive and caloric value, it is an important gradient between boiled or glucose water and milk. The milk fraction can be gradually introduced, in the slow stages previously described, and modified by the presence of the whey. The usual feed, loosely termed "whey-modified milk", contains to the pint seven ounces of whey, six ounces of boiled skimmed milk and seven ounces of water with the addition of 5% cane sugar, and when the term is used without further classification this is the mixture intended.

Whey has a caloric value of only eight Calories per ounce, and whey-modified milk a caloric value of 12·5 Calories per ounce. Thus, although such a feed is more adequate (having passed from water, through whey, to whey-modified milk), it is still not adequate even for the youngest baby. The next step, therefore, is to increase the milk fraction, thus decreasing the proportion of the other two; that is, give one part of whey, two parts of milk and one part of water, and the sugar can now be reduced to 3·8%. By the mere dropping out of the whey the feed now becomes adequate for a two or three months old infant. For older children, instead of this last step, a mixture containing one part of whey, three parts of milk and one part of water is given, the sugar content being reduced to 2·5%; the next step again is the omission

TABLE I.[1]

Age.	First Day.	Second Day.	Third Day.	Fourth Day.	Fifth Day.	Sixth Day.	Seventh Day.	Eighth Day.	Ninth Day.	Tenth Day.	Eleventh Day.
0 to 6 weeks	Glucose water.	Whey.	Whey.	Whey, modified milk.	Whey, modified milk.	Milk[2] (1), water (1), cane sugar 5%.	Milk (1), water (1), cane sugar 5%.	Milk (1), water (1), cane sugar 5%.	Milk (3), water (2), cane sugar 5%.	→	→
6 weeks to 3 months.	Glucose water.	Whey.	Whey.	Whey, modified milk.	Whey, modified milk.	Whey (1), milk (2), water (1), cane sugar 5%.	Whey (1), milk (2), water (1), cane sugar 5%.	Milk (2), water (1), cane sugar 3·8%	→	→	
3 to 6 months	Glucose water.	Whey,	Whey, modified milk.	Whey, modified milk.	Whey (1), milk (2), water (1), cane sugar 5%.	Whey (1), milk (2), water (1), cane sugar 5%.	Whey (1), milk (3), water (1), cane sugar 3·8%.	Whey (1), milk (3), water (1), cane sugar 3·8%.	Milk (3), water (1), cane sugar 2·5%.	→	→
6 months onward	Glucose water.	Whey.	Whey, modified milk.	Whey, modified milk.	Whey (1), milk (2), water (1), cane sugar 5%.	Whey (1), milk (2), water (1), cane sugar 5%.	Whey (1), milk (3), water (1), cane sugar 3·8%.	Whey (1), milk (3), water (1), cane sugar 3·8%.	Milk (3), water (1), cane sugar 2·5%.	Milk (3), water (1), cane sugar 2·5%.	Boiled skimmed milk. →
0 to 6 weeks	Glucose water.	Whey.	Whey.	Whey (2), "Vi-lactogen" 1 : 6 (1).	Whey (2), "Vi-lactogen" 1 : 6 (1).	Whey (1), "Vi-lactogen" 1 : 6 (1).	Whey (1), "Vi-lactogen" 1 : 6 (1).	Whey (1), "Vi-lactogen" 1 : 6 (2).	Whey (1), "Vi-lactogen" 1 : 6 (2).	"Vi-lactogen" 1 : 6.	→
6 weeks to 3 months.	Glucose water.	Whey.	Whey.	Whey (2), "Vi-lactogen" 1 : 4·5 (1).	Whey (2), "Vi-lactogen" 1 : 4·5 (1).	Whey (1), "Vi-lactogen" 1 : 4·5 (1).	Whey (1), "Vi-lactogen" 1 : 4·5 (1).	Whey (1), "Vi-lactogen" 1 : 4·5 (2).	Whey (1), "Vi-lactogen" 1 : 4·5 (2).	"Vi-lactogen 1 : 4·5.	→
3 to 4·5 months	Glucose water.	Whey	Whey (2), "Vi-lactogen" 1 : 4·5 (1).	Whey (2), "Vi-lactogen" 1 : 4·5 (1).	Whey (1), "Vi-lactogen" 1 : 4·5 (1).	Whey (1), "Vi-lactogen" 1 : 4·5 (1).	Whey (1), "Vi-lactogen" 1 : 4·5 (2).	Whey (1), "Vi-lactogen" 1 : 4·5 (2).	"Vi-lactogen" 1 : 4·5.	→	→
4·5 months onward.	Glucose water.	Whey.	Whey (2), "Lactogen" 1 : 4 (1).	Whey (2), "Lactogen" 1 : 4 (1).	Whey (1), "Lactogen" 1 : 4 (1).	Whey (1), "Lactogen" 1 : 4 (1).	Whey (1), "Lactogen" 1 : 4 (2).	Whey (1), "Lactogen" 1 : 4 (2).	"Lactogen" 1 : 4.	→	→

[1] After the numerals in parentheses, the word "parts" is understood.
[2] Milk = skimmed milk (boiled).

of the whey fraction to give a mixture consisting of three parts of milk, one part of water and 2·5% cane sugar, which is suitable for a five to seven and a half months old infant. Above this age the final stage for older children is to delete both water and sugar and give boiled skimmed milk. The milk should continue to be skimmed for at least two weeks after the child appears to have clinically recovered. As boiled skimmed milk has a caloric value of only 13 Calories per ounce, we frequently continue the use of 2·5% cane sugar at this stage to give 15 Calories per ounce until such time as fat can again be added to the diet. At this age extras in the form of "Farex", biscuits, apple, banana *et cetera* also give additional Calories.

The same steps can be followed when the usual food is "Vi-Lactogen", "Lactogen", "Glaxo" *et cetera* — for example, (i) whey, (ii) whey two parts, "Lactogen" one part, (iii) whey one part, "Lactogen" one part, (iv) whey one part, "Lactogen" two parts, (v) "Lactogen".

Owing to the poor nutritive value of the early stages, it is wise if possible to avoid keeping the baby too long at this level; that is, he should spend no more than twenty-four to thirty-six hours on a diet of glucose water or twenty-four to forty-eight hours on whey. However, rather longer times can be spent if necessary over the later stages—three, four or even five days if necessary. When the nutritive value of a feed is low over a long period, as has previously been mentioned, concurrent intravenous therapy with protein, serum and blood is useful in combating hypoproteinæmia and general debility, and in fact should always be given in these circumstances. During the stage of inadequate feeding infants who will tolerate amino acids orally may be given up to 15 grammes of easily absorbed protein daily by this route; this eliminates to some extent the necessity for haste during the early stages. As has also been previously mentioned, an exacerbation of the illness indicates the necessity to revert to the preceding stage or even, if it is severe, a return to the beginning (glucose water). If this is not tolerated, all administration of nutriment by mouth must cease, the gastro-intestinal tract must be given at least twenty-four hours' complete rest, and intravenous therapy is essential.

Older children, aged twelve months and over, and those accustomed to a rather sweet diet at home, may prefer the malted milk or condensed milk *régime*—glucose water followed by condensed milk 1:12, then condensed milk 1:8, then equal parts of condensed milk 1:8 and boiled skimmed milk, and finally boiled skimmed milk, which is later gradually replaced by whole cow's milk boiled. If it is preferred, malted milk may be substituted for condensed milk.

Other useful fluids to be given to older children are lemon syrup and "Vegemite" water, both of which are easily tolerated and, particularly in the latter case, readily taken. Even weak black tea will be taken when all else is refused. These mixtures should not replace the milk mixture, but may be offered as a complement when insufficient of the latter is taken.

To older infants biscuits and "Farex" can be given early (at the commencement of the third stage as a rule). It is preferred not to add apple, banana or vegetables until the infant has shown reasonable progress (usually to the "milk three parts, water one part, cane sugar 2·5%" stage), and other extras much later, though in this also the treatment must vary with the individual. Egg yolk can in most cases be given early, but should be withheld if any intolerance is shown.

For those children debilitated by a long illness the caloric value of the feeds may be increased by the use of skimmed lactic acid milk (if tolerated) or by the addition of predigested starch preparations to the ordinary mixture. Some children, particularly those in whom vomiting of part of the feed appears to have become a habit rather than a necessity, will retain feeds of thicker consistency, and here for older children baked flour preparations can be useful.

For those babies who tend to vomit or refuse the last feed of the evening and consequently are irritable and sleep little at night, a useful measure is to give a nasal feed containing a small dose of chloral hydrate (one grain); the feed is usually retained and the child sleeps well.

It is apparent that vitamin deficiency could readily occur in children receiving these diets. This must be combated by the usual methods.

The water-miscible concentrates of vitamins frequently produce vomiting in these babies, though when they are tolerated they are useful. Preparations of the vitamin *B* complex for intramuscular or oral use readily guard against vitamin *B* deficiencies. Ascorbic acid is fairly well tolerated by mouth, and when it is not tolerated an intramuscular preparation can be used. Vitamin *K* deficiency, as evidenced by a hæmorrhagic state, was pronounced in severe cases associated with liver damage, and one of the preparations of the vitamin for intramuscular use should be given regularly in any case of anything more than mild degree. Vitamins *A* and *D* present a rather more difficult problem when non-fatty preparations of these vitamins are not tolerated. However, the child is given as much sunlight as possible, and substances containing these vitamins are administered as soon as is practicable. (In this connexion it is interesting to note that intravenous wounds in these patients, though indolently showing no response to most medicaments, will frequently respond to crude cod-liver oil with "Vaseline".)

It cannot be too strongly stressed that once the infant begins to convalesce he needs interest, and to be petted and played with. Thus, once a patient begins to evince once more an interest in living, this should be vigorously fostered, but must be kept well within the tiring limit. A little petting and nursing prior to a feed at this stage will repay the time spent, as the appetite is stimulated and the child is happy and contented.

Specific Therapy.

No specific treatment has yet been forthcoming for the ordinary summer diarrhœa or non-specific gastro-enteritis. However, with the use of the dietary and intravenous therapy already described, the mortality from this disease is at present low and no deaths have occurred in the hospital for several months.

As was to be expected, in the few cases in which a specific dysenteric organism of the Flexner type was isolated, the response to the sulphonamide series of drugs was satisfactory. The *Salmonella bovis morbificans* is not susceptible to the sulphonamides or to penicillin. Four patients presenting with mixed infections of Salmonella and Flexner organisms when treated with sulphonamides soon gave negative cultural findings for the Flexner organisms, but there was no effect upon the Salmonella infection. Sensitivity tests for streptomycin on the *Salmonella bovis morbificans* did not indicate satisfactory sensitivity except in high concentration *in vitro*. However, in view of reports from overseas of satisfactory clinical use, the drug was given a trial *in vivo*.

That the Salmonella organism is capable of invading the blood stream and causing septicæmia is adequately indicated from the fact that the organism was frequently grown in culture from the liver, spleen, kidneys, brain and meninges *post mortem*. Prior to the use of streptomycin three cases of frank Salmonella meningitis were revealed on the post-mortem table. It was soon found that streptomycin rapidly controlled the septicæmic stage of the infection, and since its use no cultures of the organism have been obtained from organs other than the gut of patients given adequate therapy. In addition, several patients with pneumonia who did not respond to penicillin therapy showed rapid response when streptomycin therapy was instituted. One patient, who underwent bilateral mastoidectomy, frank pus yielding a pure culture of Salmonella group C being obtained, continued to discharge pus freely from the wounds until streptomycin treatment was commenced.

It has become our practice to institute streptomycin therapy at the earliest evidence of bacillæmia, as indicated by a developing toxæmic state. Infants aged up to three months respond satisfactorily to a dosage of 25 milligrammes every three hours, while older infants respond well to a dosage of 50 milligrammes every three hours. Dosage at this level is usually maintained for six days and control of the toxæmia is then satisfactory; however, streptomycin given by the intramuscular route has no effect upon the bowel infection. Attempts at oral administration of streptomycin have proved unsatisfactory; 0·1 gramme of streptomycin (100,000 units) with lactose in "enteric" capsules was administered every three hours to three patients without benefit or the production of negative results on cultural examination of stools. Limitation of the supply of streptomycin did not allow of further investigation into the oral route.

Complications.

During the year various phases of gastro-enteritis have been associated with different complications.

As has already been mentioned, in the early months infection of the mastoid air cavity was the prevalent complication and was demonstrated at either operation or post-mortem examination in 25% of subjects admitted to hospital (November, 1946, to May, 1947). As local evidence of infection was never present, it became customary to subject to bilateral mastoidectomy any infant who suffered repeated recurrences and for whom the prognosis appeared otherwise hopeless. In the early stages "open" ether anæsthesia was employed; but as many infants were too ill to withstand this procedure, the use of paraldehyde by intramuscular injection and local anæsthesia were found to be eminently satisfactory.

A total of 45 patients were found to have mastoid involvement, and of these 29 were subjected to mastoidectomy with 19 cures. This result must be considered satisfactory when it is considered that only infants *in extremis* were subjected to operation.

The elimination of this complication was probably due to elevation on pillows of vomiting infants; thus a ready escape route was provided for vomitus and there was a lessening of contamination of the Eustachian tubes.

Statistics reveal that during the year 45 gastro-enteritis patients also suffered from chest infections (usually bronchopneumonia), while 47 patients were found to suffer from *otitis media* during their illness. Five patients suffered from intussusception, presumably secondary to the alimentary infection. All were subjected to operation with satisfactory results.

Summary.

During the year 1947 a severe epidemic of gastro-enteritis occurred in the city and suburbs of Brisbane, necessitating admission of 384 infants to the Brisbane Children's Hospital.

About half of the cases and most of the deaths were caused by a group C Salmonella identified as *Salmonella bovis morbificans*. At the height of the epidemic the mortality rate was 40%. Successful treatment lowered this and maintained the rate at 2% to 3%. Most cases occurred among artificially fed infants. Therapeutic diets found most effective were a sequence of a 5% solution of glucose or sucrose in water, whey, whey-modified milk, modified milk.

In all severe cases intravenous treatment with half-strength physiological saline solution and 5% glucose solution was needed. Deaths occurred from protein starvation and toxic spoiling of the liver until amino acids (casein hydrolysate) were given as well. Serum therapy was of little value. In milder cases entero-colitis only occurred, but severely infected patients developed Salmonella septicæmia. Streptomycin, given in doses of 25 to 50 milligrammes every three hours by the intramuscular route, was most effective in the toxic salmonellosis stages, but had no effect on the entero-colitis.

Acknowledgements.

Our thanks are due to Dr. Alex Paterson and Dr. Felix Arden, visiting physicians, Dr. Kenneth Green, visiting ear, nose and throat surgeon, for advice and encouragement

freely given during the epidemic, and to the staff of the Queensland Institute of Medical Research for the bacteriological and epidemiological investigation of the Salmonella infection. Our thanks are also due to Dr. D. C. Fison, medical superintendent of the Brisbane Children's Hospital, for advice and for the use of hospital records, and to Miss Nancy Atkinson, of the Institute of Medical and Veterinary Science, Adelaide, for identification of the Salmonella organism.

SOME ASPECTS IN THE MANAGEMENT OF A CASE OF JAUNDICE.[1]

By MELVILLE E. CHINNER,
Adelaide.

WHEN I was asked to write a few words on the management of a case of jaundice, I felt that it would be of value to us all to renew our acquaintance with the physiology of the formation and excretion of bile, and to consider in some detail a substance of great value in the detection of liver cell disease.

Physiology of the Secretion of Bile.

Let us firstly consider the composition of bile, analysed by Hammarsten and set out by Best and Taylor.[1] Our purpose is the consideration of jaundice, which is due to the discoloration of the skin, mucous membranes, conjunctivæ *et cetera* with bile pigments. These pigments are bilirubin in the main and a small amount of biliverdin, which is an oxidative derivative of bilirubin. Bilirubin is formed from the hæmoglobin of broken-down red blood corpuscles; it is the porphyrin fraction of the hæmoglobin molecule, and it is free from both iron and globin. This pigment is produced by the reticulo-endothelial cells, which are found in bone marrow, spleen, lymph glands, liver (Kupffer cells) *et cetera*, those cells in the bone marrow being probably the most important, as it has been shown that bilirubin is formed at a normal rate in animals deprived of both liver and spleen. It may be of interest at this point to mention some facts of historical interest. As you will remember, it was in 1886 that Minkowski and Naunyn[2] considered that bile pigments were wholly formed in the liver; this supposition remained in force for some years, until McNee, working with Aschoff, discovered the reticulo-endothelial system, and showed that the only cells in the liver producing bilirubin were the relatively infrequent Kupffer cells. But Minkowski's mistake was due to the fact that his experimental animal was the goose, which houses all its reticulo-endothelial system in the liver; so removal would naturally bring about cessation of the formation of bilirubin. It is now agreed that the liver cells (except for the Kupffer cells) excrete only the bilirubin that is present in the circulating blood, and this has been shown schematically by McNee.[3] Now, once the bilirubin leaves the liver it behaves in a different way. Whilst circulating in the blood it is combined or joined with a protein or lipoid molecule; this, on account of size, probably prevents it from passing through the glomerular filter, and gives rise to the so-called indirect Van den Bergh reaction, which occurs in jaundice of hæmolytic type. But when bilirubin has once passed through the liver (possibly as sodium bilirubinate), or owing to its association with bile salts (which are conjugated in the liver), it becomes a threshold substance, and is readily excreted by the kidneys, and gives rise to the so-called prompt or direct Van den Bergh reaction. This apparently straightforward work of Van den Bergh has recently been under much discussion; the arguments centred mainly round the question whether there are two types of bilirubin or not; Watson[4] apparently demonstrated that there must be two types, and Gray,[5] more recently, tried to show that the Van den Bergh test was of no real value. Another set of observations condemning the Van den Bergh test was made and published by Corkill *et alii*[12]—namely, that the direct biphasic and delayed Van den Bergh reactions depended on the dilution of the bilirubin, and that a strong direct reaction could be made biphasic by simple dilution, owing to the fact that in concentrations of over 1·6 milligrammes of bilirubin per 100 millilitres azobilirubin does not follow Beer's law.[10] But regardless of this controversy, the direct, indirect and biphasic Van den Bergh reactions, which we all know and have used, have served a useful purpose in helping to distinguish between jaundice of obstructive, infective, toxic or hæmolytic type. The common ward tests for urinary bile have not been valuable, as they do not give a positive result until jaundice is pronounced and obvious; but more recently a modification of Harrison's test was suggested by Turner;[6] this is known as the "barium or filter strip" method, and is said to give a positive result with as little as 0·05 milligramme of bilirubin per 100 millilitres of urine. This test would appear to be simple, and may, with advantage, easily become a good ward test for bilirubin in the urine. Another substance which is of great importance in the detection of liver disease is urobilinogen, and it is capable of detection by special tests; it is the composite of two colourless chromogens—namely, mesobilirubinogen and stercobilinogen—and is formed in the intestine by the reducing action of bacteria on bilirubin. A portion of this substance is excreted in the fæces as stercobilin, but the rest is reabsorbed into the blood stream and brought to the liver again. If the liver is healthy, it reexcretes most of the stercobilin in the bile again after reforming it into bilirubin by a process of reoxidization; but a small portion remains in the blood and is excreted in the urine (provided that no great kidney damage has occurred), where it is soon oxidized into urobilin. Normally the urine contains 0·5 milligramme to 2·0 milligrammes *per centum*; but when urobilinogen is formed in excessive amounts (as in increased blood destruction—hæmolytic anæmias), or when the liver cell is damaged by infection or some toxic process, then the excess of urobilinogen cannot be reoxidized to bilirubin; so the amount in the blood increases, and this in turn is found in the urine and can be determined quantitatively if so desired. This substance, of course, disappears from the urine in cases of obstructive jaundice in a day or so, or when the bilirubin from the intestine has been cleared; but it remains present in cases of hepatitis or in cases of toxic or infective jaundice. Sometimes in the early stages of infective hepatitis it will disappear, owing to the complete obstruction to the excretion of bile, and its reappearance can be used as a prognostic point in showing that improvement has begun. This is schematically shown in a diagram given by Best and Taylor[1] or in the scheme given by Watson and Hoffbauer.[7] This test is carried out by means of Ehrlich's test: to ten millilitres of undiluted urine add one millilitre of reagent and allow the mixture to stand for three minutes; a cherry-red colour will indicate a positive reaction. This may be made more or less easily quantitative by the use of dilutions of 1:10, 1:20, 1:50, 1:100 and 1:200. Normally a pink colour may occur up to dilutions of 1:10, but any deeper colour than this is abnormal and a positive result in increasing dilutions would point to progressive disease. Pollak[10] has put these tests very concisely. After mixing the urine and reagent, wait for three to five minutes; normal urine will show a faint reddish tinge, which can be intensified by heating; it is said that normal urine in dilutions above 1:10, and cold, will not give any red colour.

> For practical clinical purposes it is usually sufficient to know whether urobilinogen is *absent* from the urine (no red colour develops even on heating), present in *normal* amounts (faint pink in the cold), or present in *increased* amounts (distinct red colour in cold urine). In the latter case the degree of the increase may be judged by serial dilution.

Classification of Jaundice.

At this point we must leave the discussion of the physiological and normal, and confine ourselves to a state in which increased bilirubin is present in the blood, and hence a state of jaundice exists. It now becomes necessary to attempt to classify jaundice. Rich proposed the terms

[1] Read at a meeting of the South Australian Branch of the British Medical Association on February 26, 1948.

"retentive and resorptive" jaundice; but I myself find McNee's classification easier to work with and more clinically descriptive: (i) obstructive, (ii) toxic or infective, (iii) hæmolytic.

It is, of course, of the utmost importance to know which variety of jaundice is present, as the treatment may be either medical or surgical. In 1916 Van den Bergh rediscovered the use of Ehrlich's diazo-reaction, and he thought that this would be of vital import in the differentiation of jaundice; but owing to the fact that rarely is only one defect present at one time—a direct test will always give an indirect result; and the reaction appears to depend on the amount of bilirubin present in the blood—this reaction becomes unreliable except perhaps in the case of pure hæmolytic jaundice. Clinically, many factors will aid in differentiating between the obstructive and hæmolytic types; for example, in the former the skin and conjunctivæ are usually much more yellow and bilirubin appears in the urine very early, whereas in the hæmolytic varieties the urine does not contain bilirubin, for the postulated reasons as given earlier. In the obstructive variety the icteric index is much higher, and the test for urobilinogen may soon give negative results. Also in the obstructive variety there is evidence of resorption of other bile constituents into the blood—for example, the bradycardia and pruritus which are thought to be due to the bile salts; also the blood has a reduced coagulability due to the lack of prothrombin, as the jaundice causes a lack in the absorption of vitamin *K*. Then also in the hæmolytic varieties various tests of the blood will reveal some abnormality in most instances. Generally it is not difficult to distinguish between the obstructive and hæmolytic types of jaundice, but it is not at all easy at times to separate the obstructive from the infective or toxic types. It is at this point that liver function tests must be called for, and we soon find that many tests must be made, as the liver performs so many functions that one test is in no way conclusive of liver damage; so various tests must be performed to show defect in any of the various functions of the liver, and these results correlated with the clinical findings. Let us now consider some of the liver functions that are capable of being subjected to test. I shall mention six different liver functions that are capable of being tested to some degree.

Functions Concerning Bile Pigment Excretion.

Bile pigment excretion can be estimated by the icteric index, which can give the measure of bilirubin in the blood. Its weakness is that other blood pigments—for example, carotin—may be present. It is better to use the quantitative Van den Bergh test. Also a positive response to the test for urobilinogen reveals a defect in the reexcretion of this substance except in cases of excessive hæmolysis, with resultant excessive production of bilirubin; but this is rare, as the liver has apparently enough reserve power, if healthy, to reexcrete almost any amount; hence a positive response to the urobilinogen test almost always points to a sick liver cell.

Hæmatopoietic Functions.

Mainly tests of hæmatopoietic functions concern estimations of the blood albumin and globulin contents; in almost all cases of liver cell destruction there is an upset in the albumin-globulin ratio. This is due to the fact that these substances are thought most probably to be elaborated in the liver. Arising out of this altered ratio and from the excess of γ globulins in the blood, come the serum colloidal gold flocculation and turbidity tests. I may quote MacLagan[11] at this point:

> Thus electrophoretically separated serum gamma globulin acts as the precipitating agent, and an absolute increase in this fraction has been demonstrated in hepatitis. Similarly, normal serum albumin acts as an inhibitor, and this fraction is often reduced in hepatitis; moreover, hepatitis albumin is less effective as an inhibitor than normal albumin.

These tests do not accurately test any liver function, but they produce positive results in varying degrees when the liver cell is sick. Generally these flocculation and turbidity tests give positive results in hepatocellular jaundice and negative results in obstructive jaundice. But fallacies can arise, as the amount of secondary cellular liver damage in obstructive jaundice varies directly with the length of time of the obstruction, but this rule breaks down if infection is present; then cellular damage occurs much earlier, and so these tests will produce positive results early, and you may be misled, especially if these are the only tests performed and the case history *et cetera* were not carefully considered. Another carbohydrate-containing protein, called prothrombin, is mainly produced in the liver (there is a little evidence that bone marrow may produce some), its production being governed by vitamin *K*; so estimations of prothrombin time, and the response to injections of vitamin *K*, may help to separate surgical from medical jaundice. When bile is absent from the intestine vitamin *K* is not absorbed, and so the prothrombin time is lowered; if you give vitamin *K* parenterally, then in a few hours the prothrombin time will be restored to normal, provided that the liver cell is healthy. But if the disorder is hepato-cellular jaundice due to damaged liver cells, then the giving of vitamin *K* will make no or little difference to the prothrombin time. Fibrinogen is also thought to be manufactured in the liver, but this is difficult to estimate, I believe, and so it is not used in liver function tests.

Effects of Liver Cell Damage on Carbohydrate Metabolism.

We have used for years two sugars, lævulose and galactose, testing the effect of liver cell damage or carbohydrate metabolism. Latterly mainly galactose has been used, as it is a sugar which the liver can metabolize freely in the face of obstructive jaundice of months' duration, but not in the face of hepato-cellular jaundice; no galactose can be changed to glucose except by the liver, but some lævulose is changed on absorption, and so the latter sugar is less reliable. Probably the best of these tests is the intravenous galactose test which was recently so ably sponsored by Althausen;[8] in this test the variability in the rate on intestinal absorption is eliminated, and the result depends on the amount of galactose left in the blood at the termination of the test.[9]

Test of Detoxication.

The function of detoxication is thought to be tested by the estimation of hippuric acid in the urine after the giving of a test dose of sodium benzoate; a certain amount of hippuric acid must be recovered from the urine, and if less than a certain minimum is obtained, then some liver cell disease is present. But this test has several parts, and it would appear that glycine is necessary for this change to take place; glycine is said to be formed in the liver, but the kidney tubules are considered to be the site of the manufacture of hippuric acid.[10] Hence this test as a liver function test is really only a test of the capacity of the liver to synthesize glycine.

Power of the Liver to Excrete Foreign Substances.

In tests of the power of the liver to excrete foreign substances, the clearance of bromsulphalein from the blood within a certain time is looked upon as a valuable test of the integrity of the liver cell. This probably is the most sensitive of all tests of liver failure in the absence of jaundice, and is used as a method of prognosis before surgery.

Empirical Tests.

The estimation of the serum alkaline phosphatase content is useful in the absence of bone disease—for example, Paget's disease, hyperparathyreoidism *et cetera*. A reading of over 30 units is found in jaundice of obstructive origin, and a reading of under 30 units or a normal reading in jaundice of parenchymatous origin. In the presence of jaundice it is thought that lack of absorption of another fat-soluble vitamin—vitamin *D*—may be responsible for the result of this test. So it can be gathered that in order to test liver functions reasonably accurately, many tests have to be done, and the results taken in conjunction with clinical data.

Report of a Case.

Perhaps the quoting of a case may help to fix in our minds something of the procedure necessary to separate obstructive from parenchymatous jaundice.

Recently I examined a very stout woman, aged forty-one years, who gave a history of shivering and a rise in temperature six months before. This was followed by a period of indifferent health; three months later she developed jaundice associated with a rise in temperature, the appearance of ascites and many skin telangiectases, and a tendency to hæmorrhage from the gums. She gave no history of any attacks of gall-stone colic, nor any suggestion that she had been previously jaundiced; there was a history of moderate alcoholism.

The patient was seen to be a stout woman, moderately jaundiced. Numerous spider-like telangiectases were present over the face, the neck, the thorax and the upper parts of the arms, and the gums were bleeding round the teeth margins. There was free fluid in the abdomen, and the urine was found to contain a little bile by the iodine test, but yielded a strongly positive result to the test for urobilinogen.

Here the problem was whether this woman was suffering from obstructive jaundice due to stones, neoplasm *et cetera* blocking the common bile duct, or from hepatogenous jaundice due to subacute hepatitis. The woman's age and obesity were in favour of a diagnosis of obstructive jaundice, but there was no history of biliary colic or prolonged indigestion, and no loss of weight had occurred. Also the jaundice was of six weeks' duration, but was not becoming progressively deeper; this was against a progressing obstructive lesion. The presence of a large amount of urobilinogen in the urine so many weeks after the onset of the jaundice is of great importance; in obstructive jaundice there may be an increase in urobilinogen for a few days, but as soon as no further bile reaches the intestine, all the urobilinogen is excreted, and so no more appears in the urine. Thus in this case the continued presence of urobilinogen pointed to an hepatogenous cause of the jaundice. The tendency to hæmorrhage was due to a prothrombin deficiency, the bleeding time was prolonged and the prothrombin time was below normal.

The patient was given vitamin *K* parenterally without any sign of relief of the hæmorrhage. This fact again pointed to the liver cell as the source of the trouble, as in obstructive jaundice the giving of vitamin *K* rapidly improves the prothrombin time, but in the presence of liver cell damage it is either very slow in effecting an improvement, or else none occurs at all. The diagnosis in this case was considered to be subacute hepatitis, probably a recrudescence of chronic hepatitis. This proved to be the case, as the patient rapidly went into a cholæmic state and died. On post-mortem examination her liver was very small, yellowish-green in colour, and studded with small knobby areas amongst the fibrous contracted portions, the picture being one of subacute yellow atrophy superimposed on a more chronic hepatitis.

Owing to the patient's rapid deterioration and to the short time that she was under my care, no further liver function tests were performed. However, I consider the constant presence of urobilinogen and the failure to respond to vitamin *K* as very valuable tests, which we can all have carried out reasonably easily and cheaply. I would make the suggestion that in addition to the above-mentioned tests a serum colloidal gold test should be performed. This will produce a strongly positive result in hepatogenous jaundice and a negative result in obstructive jaundice. A serum phosphatase test is also useful; this will produce a reading of over 30 units in the obstructive type of jaundice and of less than 30 units in the hepatogenous type. If good laboratory assistance is readily available, other tests may be carried out as a routine measure; these were recently summarized by Snell.[12] I believe that the most valuable tests for differentiating obstructive and hepato-cellular jaundice are (i) the urinary urobilinogen test, (ii) the serum colloidal gold test, (iii) the serum alkaline phosphatase test, and (iv) the response of the plasma prothrombin time to vitamin *K*.

Another method of diagnosis that has been used of recent years is that of liver biopsy. This operation aims to obtain a small section of liver tissue by means of a hollow cutting needle introduced into the liver, and this, on withdrawal, provides a small cylinder of liver tissue. I would not recommend this procedure to the inexperienced, as liver tissue can bleed viciously, and if the patient is jaundiced, then is the tendency to even more severe hæmorrhage. The test must be performed only when the prothrombin time is about normal, the patient must cooperate with regard to breathing, and the operator must have had practice; but this method will frequently give valuable help in the assessment of subacute hepatitis—the presence of advancing cirrhosis and at times of secondary carcinoma may be proved.

Conclusion.

Finally, I shall make a few remarks on the dietary treatment of the jaundiced patient suffering from hepatitis in its varying stages. The first requirement is rest, and this must be maintained until the patient has completely recovered. In the acute stages this is not difficult, as the patient frequently feels very ill; but in the more chronic forms of hepatitis it is easily proved that exercise will bring about a swelling of the liver and exacerbation of symptoms, and when the patient is walking about avoidance of fatigue is essential. The question of Calories and diet is also of importance. In the acute stages probably the patient will take only fluids with a high carbohydrate content; if the vomiting is severe, then adequate fluids with glucose must be given by the intravenous drip method. But as the hepatitis becomes subacute, a high Calorie diet becomes necessary. We have in the past always thought that diets poor in or free from fat were a necessity; but recent opinion as stated by Chester Jones[15] points out that such diets are undesirable, as they are poor in Calories and unpalatable. Recently I have been using gradually increasing amounts of fat in these patients' diets. Snell advises 350 to 500 grammes of carbohydrate, 100 to 150 grammes of protein, and 100 to 150 grammes of fat. It was also considered a few months back that diets very rich in protein, containing first-class proteins with high cystine and methionine contents, were necessary, with the addition of other lipotropic factors such as choline and inositol; but Jones has also suggested that these substances may be necessary to the experimental animal, but not in diets that are well balanced. I have also used large amounts of the vitamin *B* group, crude liver injections and dried brewer's yeast; I would still recommend the use of these substances in chronic hepatitis until we have more evidence for or against them. Sometimes the patient suffering from subacute hepatitis may become œdematous and exhibit ascites. This is due to the upset in the serum proteins—the contents are usually low and the albumin-globulin ratio is upset; this condition is best treated by a diet poor in or free from salt and with a high protein content, or probably by the intravenous administration of blood albumin. Jones states that if the serum protein level is below three grammes *per centum*, then 50 grammes of human albumin should be given intravenously every day for a week. In the presence of normal serum protein levels the use of mercurial diuretics may be advantageous.

I again stress the need of a thorough clinical examination and the use of the laboratory tests as adjuncts, and urge you to remember that the liver has enormous reserves, and that often much damage has to be done to liver cells before positive test results are obtained. Harrison has recently written that at the best, liver function tests are not wholly satisfactory; one American speaker said that the great advantage of liver function tests was that they took so long to perform and that by the time the results were obtained the condition could be diagnosed clinically. But I believe that if they are used properly a great deal of valuable information can be obtained from the tests that have been mentioned.

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Reviews.

PEACE OF MIND.

"PEACE OF MIND" is written by Rabbi J. L. Liebman in the belief that "social peace can never be permanently achieved so long as individuals engage in civil war with themselves".[1]

The author is a Jewish professor of philosophy and a distinguished radio preacher. He has a profound respect for psychiatry and believes that theology must learn the tolerance of psychiatric wisdom if it is to be "a true minister to our civilization and its discontent".

The book, though written by a layman, is based on sound psychological principles, and it is apparent that there has been considerable assistance from psychiatrists. It aims to assist the individual man in his conflicts and worries, and to help him in the creation of tolerance which is essential to world brotherhood and peace.

Rabbi Liebman writes as a Jew and draws much material from the Hebrew prophets. It is interesting to note the psychological implication. He claims that the Jewish religion intuitively understands the part played by emotion. Spectacular holidays and festivals of New Year, Atonement, Harvest, Purim and Passover, serve to abreact feelings and purge the inner life. It is an aspect which goes some way to explain the persistence of the Jewish race.

As might be expected, the author becomes involved in theological arguments. He realizes that by espousing the aid of psychiatry in the understanding of God there must be criticism. He takes the broad viewpoint. His commonsense attitude is shown by his understanding of the infantile viewpoint held by many worshippers. "To them the Lord is a master chef at a gigantic fish fry—a cosmic bellhop who should respond to their every summons." With such an outlook his views on the way to a "peace of mind" for the average man could be expected to be interesting. They are not disappointing.

On the matter of conscience which makes cowards, he gives examples and shows that the solution lies in understanding of the true roots of the anti-social conduct. We must "view ourselves neither as naughty children nor as spotless angels, but as mature men and women with all our mortal imperfections on our heads".

On the importance of identification the Rabbi is on sound ground. "In a spiritual sense, we digest our heroes and heroines and make their way of life part of our own emotional substance. For man, of all the creatures on earth, has the greatest power of imitation and identification with others." On this basis we need love in our childhood if we are to have a world of happy and tolerant people.

The author describes some graphic examples of morbid fear and grief due to a wrong psychological handling. He points out that because a three-year-old child, who lost her mother, was not told the truth, her grief symptoms persisted into adulthood!

In the forefront of an excellent chapter on immortality, designed to soften the fear of dying, there is a quotation from Montaigne. He advises us that "like a full-fed guest, depart to rest. . . . Yield your torch to others as in a race".

Professor Liebman discusses the causes of atheism and finds that frequently they are the result of frustration in childhood. Some individuals are repressed, ashamed of emotion and turn to objective science as an escape.

The author claims that religion should be able to liberate man from the slavery of his misconceptions and emotional shackles. There are many—Jew and Gentile—who will be helped by a perusal of this well-written book. It can be strongly recommended to medical practitioners who consider that lay advice with a religious bias is indicated for a personality problem.

[1] "Peace of Mind", by Joshua Loth Liebman; 1947. London and Toronto: William Heinemann, Limited. 8" x 5¼", pp. 220. Price: 8s. 6d.

A YEAR BOOK OF DERMATOLOGY AND SYPHILOLOGY.

MARION B. SULZBERGER AND RUDOLF L. BAER, editors of "The 1947 Year Book of Dermatology and Syphilology" (and of many preceding volumes in the series), introduce the book with a vigorous and convincing apologia for dermatology and the dermatologist.[1] Under the title "Some Common Misconceptions Regarding Dermatology" they set themselves to dispose of some of the alleged skeletons in the specialist's family cupboard, such as the reputation for the use of unusually strange names for skin diseases to cover up ignorance or laziness, and the idea that dermatologists are unscientific in treatment and have contributed little to progress in medicine. They also decently inter the hoary jest that dermatologists have " a wonderful specialty"—their patients "never get cured and never die"—and dispel the illusion that it takes only "a minute" to complete the average dermatological consultation. There are some wise words on the exaggerated reputation of skin diseases for contagious qualities, and the article concludes with an unanswerable argument that most of the common current misconceptions cannot be blamed on to the dermatologist, but are the result of faulty understanding of the specialty by the general medical world and by the laity. The remedy lies in better hospital, laboratory and research facilities for the dermatologist and adequate education of the medical undergraduate and graduate concerning the specialty. This article should be read by every medical practitioner and especially by every teacher.

The rest of the book also warrants the attention of quite a wide medical public, but especially of the general practitioner and, of course, the dermatologist. An extensive medical literature is covered, mostly from North America and England with occasional references to continental journals. Two papers by Australians are included: one by A. T. H. Jolly on the effect of intravenously administered antimony compounds on pyogenic skin lesions, and one by J. P. O'Brien on *miliaria rubra*, tropical anhydrosis and anhydrotic asthenia. A large opening section on the treatment and prevention of skin diseases is followed by sections on physical therapy, on eczema, urticaria and allergy, on drug eruptions, on miscellaneous hæmatogenous dermatoses and on other dermatoses, on cancers and other tumours, on *mycosis fungoides* and leuchæmia, on fungous infections, and on other infections and infestations. The section on venereal diseases is concerned mainly with syphilis, treatment with penicillin and recent investigations of serological tests having pride of place; the rest of this section deals mostly with *granuloma inguinale* with particular reference to streptomycin treatment. The volume concludes with a section on investigative studies and a miscellaneous section in which the editors offer a somewhat sceptical reception to a group of papers on psychological factors in skin diseases. Throughout the book editorial comment is offered freely and adds greatly to the value of the material summarized. All who are interested in dermatology and syphilology will find this volume invaluable.

[1] "The 1947 Year Book of Dermatology and Syphilology", edited by Marion B. Sulzberger, M.D., and Rudolf L. Baer, M.D.; 1948. Chicago: The Year Book Publishers Incorporated. 7" x 4⅞", pp. 604. with many illustrations. Price: $3.75.

The Medical Journal of Australia

SATURDAY, JULY 17, 1948.

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THE RESEARCH ACTIVITIES OF THE NATIONAL HEALTH AND MEDICAL RESEARCH COUNCIL.

THE National Health and Medical Research Council held its twenty-fourth session at Sydney on November 12, 1947, and a report has at length been printed. The document is little more than a skeleton report, but this fact is not without value. It is the skeleton of the Council, the framework on which it is built and the supports to the framework which are of importance. These may perhaps be more easily discerned in a bare document than in one complicated by habiliments of flesh and surface decorations. On previous occasions attention has been drawn to the disability under which the Council labours, because its dual function is indicated by its name. However that may be, we know that by delegating special aspects of its work to special committees to which non-members have been coopted, the Council has done what it can to overcome this disability. At its November meeting the Council generally allots its research grants for the coming year, and the present report contains a list of the grants for 1948. The Council makes its grants after consideration of the recommendations of the Medical Research Advisory Committee. This committee comprises the following persons: Professor H. R. Dew (chairman), Dr. H. J. Ritchie, Dr. W. F. Simmons, Professor W. L. Mitchell, the Commonwealth Director-General of Health, Professor F. M. Burnet, Professor H. K. Ward, Professor R. D. Wright, Dr. E. V. Keogh and Dr. R. E. Richards (medical recorder). Dr. W. F. Simmons, who is the representative on the Council of the Federal Council of the British Medical Association in Australia, in his report to the March meeting of the latter body, stated that when the committee met before the November meeting of the National Health and Medical Research Council it found that applications had been received for research grants totalling £57,746 10s. The available grant of the Government for research was £50,000. In its recommendations therefore the committee was compelled to refuse assistance to many new applicants and also to curtail work in some established projects. It is impossible in the available space to give a list of grants made by the Council; nor is this necessary. It should, however, be noted that the sum of £1000 has been granted to each of the Universities of Melbourne, Sydney, Queensland and Adelaide for the purpose of training personnel for medical research. This, of course, is commendable, but the amount of the grant in each instance seems inadequate. The Council probably believes, and would be justified in the belief, that no larger grant for this purpose should be made until it has at its disposal the large and permanent endowment that it should have. The grants made are set out under the following headings: Bacteriology, Biochemistry, Biophysics, Clinical Research Unit, Dentistry, Medical Survey, Neurology, Neurophysiology, Nutrition, Obstetrics and Gynæcology, Birth Rate Committee, Physiology and Pharmacology, Tuberculosis, Virus and Publications. It is interesting to note that the Council continues its grant to *The Australian Journal of Experimental Biology and Medical Science*. It pays thirty shillings per page for the publication of work submitted by persons receiving grants from the Council, up to a maximum of £75. Commendable also is a grant of £750 for fares and travelling expenses to enable a worker to visit the United States of America for eight months. What must surely be a precedent is a grant of £205 to another individual for travelling expenses to attend the International Congress of Pediatrics at New York.

The practical point that emerges from a consideration of the facts stated and from a perusal of the grants made is the need for the allocation of more money for research. What is needed is a permanent grant-in-aid such as is made to the Medical Research Council in Great Britain by the British Government. But governments are unwilling, or make the excuse that they do not wish, to pledge to expenditure for such a matter any administration that may succeed them in another Parliament. Under the present arrangement research is carried on from year to year by a kind of hand-to-mouth existence. No worker paid by the National Health and Medical Research Council has any feeling of security in the continuity of his work, to say nothing of any superannuation after a precarious and possibly long life. Australia cannot hope to retain her research workers in these circumstances. The Council approved a recommendation of its Medical Research Advisory Committee that Cabinet should be asked to provide more funds for research work. It decided that the request should be prepared under the following main headings: (*a*) A statement showing the present position in regard to funds for projected developments. (*b*) Travelling fellowships (the possibility that these may no longer be available from overseas). (*c*) The placing of Fellows who are returning from abroad. (*d*) New developments of the future in all capitals and the desirability of granting fellowships to worthy applicants. The Council also decided to ask the Minister to introduce to the Prime Minister a deputation to present the case for increase in the funds for medical research in Australia. Another important decision arose from a discussion on the fixing of salaries and travelling allowances for research workers. Here it may be pointed out that not only are the salaries paid to some medical graduates inadequate, but those of science graduates engaged in research are often scandalously and absurdly low. The Council decided to direct the Medical Research Advisory Committee to review

all salaries of research workers and to draw up a scale of payment.

The Government may possibly be induced to do something in regard to the provision of more funds for research because of another circumstance. This is the bringing into being of the Australian National University which was discussed in last week's issue. This institution will be concerned chiefly with research in physical sciences, medical research, social sciences and Pacific studies. As Professor Copland stated in his address at the "commencement" function at the University of Melbourne, the new university will draw on the resources of the existing universities, as regards both staff and students. Even if this process of attraction or appropriation should be extensive, all research will not be centred at Canberra. In other words the National University will be complementary to existing universities in the field of research. Professor Copland states that the monetary resources of the National University are ample. It would be unreasonable to suppose that the Government would be generous to one of its research agencies and wilfully parsimonious to another. The obvious course for adoption is the creation of an Australian Medical Research Council, charged with the oversight of all government-aided research in the Commonwealth and with this as its only responsibility. Such a national undertaking should be put on a permanent basis so that long-range projects could be undertaken by workers freed from economic anxieties and from the need to look overseas for security. All those who have to do with research in this country should ask themselves whether the present is not the most suitable time to press towards this goal.

Current Comment.

DIPHTHERIA CARRIERS TREATED WITH PENICILLIN.

DIPHTHERIA CARRIERS have long been a trial alike to themselves and to their medical attendants. Many forms of treatment have been devised, all of which may be tried in certain cases with depressingly persistent failure. The advent of penicillin offered new possibilities as it was early found that *Corynebacterium diphtheriæ* was susceptible to penicillin *in vitro*. However, the parenteral administration of penicillin met with little success, presumably because of the superficial location of the organisms in the carrier's throat or nose. Attention was then turned to local administration of the antibiotic with much more encouraging results. R. A. Kocher and W. J. Siemsen[1] treated with intramuscular injections of penicillin nine patients who had recovered from clinical diphtheria, but in no case were able to rid the patients' throats of the organism. Then, on the personal advice of Sir Alexander Fleming, they had lozenges prepared, each containing 1000 units of penicillin, and administered one to 31 carriers every hour for twelve doses during waking hours over a period ranging from three to twelve days. A spray containing 1000 units of penicillin per millilitre was applied to the nose. Twenty-three patients were promptly freed of organisms, seven others were immediately freed by tonsillectomy and the remaining carrier responded to further local treatment after tonsillectomy. A. J. Levy[2] has pointed out that Kocher and Siemsen did not distinguish between virulent and avirulent organisms, and in his own investigations has eliminated the carriers of avirulent organisms. He records the treatment of three carriers who had virulent *Corynebacterium diphtheriæ* in their pharynxes by means of penicillin lozenges (1000 units in each) administered every two hours for six days, and of one carrier with organisms in both nose and pharynx by means of two millilitres of penicillin solution (containing 10,000 units per millilitre) administered by spraying with an atomizer every two hours for six days. In all cases treatment was immediately and permanently effective. Levy draws attention to the fact that he used a solution for spraying of 10,000 units per millilitre as against 1000 units per millilitre used by Kocher and Siemsen and other investigators. Levy's series of cases is, of course, very small and the series of Kocher and Siemsen is not large; there are very few other references to the treatment in current literature. It is difficult to believe, however, that others have not thought of this rather obvious method of treatment, so that the absence of unfavourable reports may perhaps be taken as supporting these limited but rather convincing reports. It is to be hoped that others will make their results known in the common interest.

[1] *Annals of Internal Medicine*, May, 1946.
[2] *The Journal of the American Medical Association*, March 27, 1948.

TUBERCULOSIS IN THE BRITISH ZONE OF GERMANY.

THE news from the British Zone of Germany is not all doleful. We hear so much about political differences among the four great Powers and of food shortages among the German people that there is a certain relief in reading a recent publication from the British Foreign Office.[1] An inquiry has been made into tuberculosis in British-occupied Germany by M. Daniels and P. D'Arcy Hart, who are members of the scientific staff of the Medical Research Council of Great Britain. The inquiry was made at the request of the Public Health Adviser to the British Military Governor in Germany. Daniels and Hart's terms of reference were as follows: (*a*) to review the notifications for pulmonary tuberculosis in the British Zone and the British sector of Berlin, and to determine how far they represent a real or paper increase in the incidence; (*b*) to remark on the method of notification now used; (*c*) to make recommendations to improve the general system of notification, if this is found to be unsatisfactory; (*d*) at discretion to make any inquiries, recommendations or criticisms about the general position of tuberculosis. Unfortunately Daniels and Hart had very little time for their investigation—September 24 to October 6—and so they concentrated more particularly on the first three of their terms of reference. In spite of this, they record some interesting observations. They had access to both British and German records, visited tuberculosis dispensaries and also "good" and "bad" homes.

The findings in the area of the country controlled by Britain were different from those obtained in the British sector of Berlin. The war-time rise in mortality from pulmonary tuberculosis in the British Zone has been checked and the death rate is now more or less stationary. The mortality from non-pulmonary tuberculosis is still increasing in some parts, but these deaths were found to account for only one seventh of the total tuberculosis deaths. The present levels of tuberculosis mortality in the zone are 51 to 85 per 100,000 deaths and these, we read, compare favourably with the rates in parts of Great Britain and other European countries. Turning to the British sector of Berlin, we find that the increase in tuberculosis mortality which was much greater during the war than in the British Zone, has still a much higher level "and constitutes a grave problem". However, the war-time rise has been checked and the mortality from pulmonary tuberculosis seems to be declining. In the British sector of Berlin also the mortality from non-pulmonary tuberculosis is at present stationary. Daniels and Hart seek to discover why the war-time rise in

[1] Tuberculosis in the British Zone of Germany, with a Section on Berlin. Report of an Inquiry made in September-October, 1947, by M. Daniels, M.D., D.P.H., and P. D'Arcy Hart, M.D., F.R.C.P. The Foreign Office; 1948. London: His Majesty's Stationery Office. 9½" × 6", pp. 32. Price 6d. net.

pulmonary tuberculosis in the British Zone has been checked and why the present position is no worse than it is. One important factor in their opinion is institutional accommodation, which is generous as judged by standards in Great Britain and other parts of Europe. In the British Zone of Germany there is a ratio of 2·2 beds to every death from tuberculosis; in Great Britain the figure is 1·3. Another reason is thought to be the provision of supplementary rations for the tuberculous. Living conditions are also expected to have influenced tuberculosis trends; Daniels and Hart state that at the time they wrote expert evidence suggested that the deterioration accompanying the end of hostilities in Germany had been checked. They point out that information obtained in the autumn of 1947 from qualified Allied observers is that the state of nutrition of the population has not worsened and that in some groups it has improved. Another fact mentioned is that other mortality statistics known to be sensitive to social conditions have recently shown a more favourable course. Both the general death rate and the infantile mortality rate have been lower in 1947 than in 1946. The increase in non-pulmonary tuberculosis finds a ready explanation in the high incidence of tuberculosis in German cattle and in the post-war deterioration of pasteurization plants with scarcity of fuel to keep going those that are fit for use. One fact covering the whole subject and described as regrettable is that German officials, some of them non-medical, have repeatedly supplied to Allied journalists and other visitors misleading and sometimes false information about the tuberculosis situation in Germany. This has had the effect of putting tuberculosis in Germany, without justification, on the level of a sensational news item.

In discussing the tuberculosis picture in Berlin, Daniels and Hart point out that the mortality in the city, which was higher than in the rest of Germany, rose alarmingly, as already stated, during the war. The present high level is thought to have a probable causative factor in the peculiar distribution of in-patient hospital accommodation. In 1939 four-fifths of Berlin's beds for tuberculosis patients were in other parts of Germany; at present the bed ratio is 0·7 bed for each tuberculosis death. Daniels and Hart mention other reasons given for the present tuberculosis picture of Berlin and admit that the feeding disadvantages of Berlin citizens may have been partly responsible.

In this short survey mention has not been made of parts of the report dealing with dispensary case registrations, suggestions for improving the reporting of new tuberculosis cases and mass radiography figures. These will probably be studied by experts in the subject. Our object has been to show that there are some encouraging aspects in life as it is lived by Germans under British control.

THE CRISES OF CONGENITAL HÆMOLYTIC JAUNDICE.

ALTHOUGH congenital hæmolytic jaundice is not a common disease, the alarming blood crises which occur in its subjects are so striking that they perhaps loom more in the memory of physicians who have treated them than their frequency would suggest. It is a clinical truism that the people suffering from this malady look much more ill than they are, for they may maintain fair health in spite of a chronic depression of the blood state, accompanied by a characteristic complexion. The suddenness with which one of these blood crises may occur is astonishing, and the blood count may drop alarmingly in a few days. Perhaps it has not occurred to those interested in this disease that there is a similarity between these crises and those which occur in other conditions recognized to be aplastic in mechanism. Paul A. Owren has published an investigation into the blood crises occurring in six persons suffering from chronic hæmolytic jaundice.[1] Four of these were members of the same family, and they became ill within a few days. One of the remaining patients came of a family of particular historical interest, as three of its members had their condition described in the literature as acute pernicious anæmia before Minkowsky described this congenital disease. All the patients conformed to the criteria required for diagnosis—jaundice without bilirubin in the urine, anæmia, enlargement of the spleen, increased fragility of the red cells, increase in reticulocytes in the blood, and the presence of spherocytes.

In every instance the crisis began with a sudden rise in temperature; two patients had rigors. Army medical officers who served in malarious areas may perhaps be reminded of blackwater fever. The temperature remained raised for about ten days till the crisis was over. Owren could not demonstrate any increase in the size of the spleen during the crisis, nor any deepening of the jaundice.

One of the patients, a child, accompanied his father when the latter was sent to hospital with a hæmolytic crisis, and, though not ill at the time, was observed to be a sufferer from the same disease, and was at once examined. Eight days later he had a sudden crisis himself, and thus afforded an opportunity for observation before and during a crisis. During the phase of acute anæmia in these patients the chief findings were reduction of blood cells and hæmoglobin to about half their usual value and complete disappearance of reticulocytes. Examination of the bone marrow showed that this was accompanied by a complete cessation of erythropoietic function. When recovery began active production of precursor red cells was observed. The rate of regeneration was such that the erythroblasts, which had been reduced to about 3000 per cubic millimetre in the bone marrow, increased rapidly to about 300,000 per cubic millimetre. The pictures presented by the marrow were striking, the usual dominance of erythropoiesis yielding to an almost exclusive prominence of myelocytes and promyelocytes. The cells from which regeneration of the red cells was accomplished were large single cells, with basophilic protoplasm. These observations show that during a crisis an aplastic state of the bone marrow exists. The author remarks that the spontaneous recovery shows a fantastic ability of the erythron to regenerate. This recuperative capacity of the bone marrow is remarked in other aplastic states, and is one of the reasons why it is so hard to assess the value of any treatment in these conditions. The point that now arises is whether this aplasia of the bone marrow is the cause of the crisis or whether there is also an increase in hæmolysis. The latter has been thought to be the cause by most workers in the field. Owren points out, however, that there was no evidence of increased hæmolysis in his series; on the contrary he found that there was a decrease in blood-derived pigment both in the serum and in the urine during the crisis. He also thinks that, as bilirubin production is at the time decreased, the observations previously made of deepening of the jaundice were incorrect. At the same time, he remarks that if the production of red cells is suddenly suspended, the speed with which anæmia will be produced will depend upon the lifetime of the red cells. Unpublished data collected by the author show that red cells from a patient with congenital hæmolytic anæmia when transfused to a normal person have a maximal lifetime of about fifteen days, whereas normal red cells transfused to a patient with hæmolytic jaundice live about 110 to 120 days. If an average lifetime of fifteen days for red cells in these patients may be assumed, calculation shows that the rate of drop of red cells in the blood following a cessation of marrow activity would correspond with the observed findings. Owren believes that the fundamental defect in this disease is in the red cells, and not in the reticulo-endothelial system, and that there is no evidence that hæmolytic processes are more active during a blood crisis. If this is so, the crisis of this disease is another of those crises affecting the bone marrow which are often so obscure.

Growing knowledge of the toxic action of some drugs on the blood-forming tissues has shed a good deal of light on such serious emergencies, but the mechanism is not understood. There seems no harm in calling such accidents of the marrow "cryptogenic", provided that we do not think that this salves our scientific conscience.

[1] *Blood*, March, 1948.

Abstracts from Medical Literature.

DERMATOLOGY.

Skin Sensitivity to Penicillin Preparations.

R. H. MEARA (*The British Journal of Dermatology and Syphilis*, January, 1948) reports three cases of skin sensitivity to penicillin preparations in subjects of exogenous dermatitis in order to illustrate the importance of analysing reactions to compound applications. In all three cases penicillin as such was found not to be the noxa concerned. The author states that skin sensitivity to topically applied penicillin itself has been extremely rare in University College Hospital Clinic. In the first case of his series skin sensitivity to penicillin existed only when penicillin was incorporated in the cream base described. In the second and third cases penicillin was not concerned at all, the responsible agents being wool alcohols and chlorocresol respectively.

Liquid Oxygen in Dermatological Practice.

R. L. KILE AND A. L. WELSH (*Archives of Dermatology and Syphilology*, January, 1948) have used liquid oxygen over a period of years and have observed it to be effective in more than a thousand dermatological cases. Applications are made by using cotton wrapped round a wooden applicator, the size and shape of the cotton tip being varied with the size of the lesion to be treated. The applicator is readily dipped in the oxygen, withdrawn and applied to the lesion, with varying amounts of pressure. As with carbon dioxide, the firmer the pressure, the deeper the effect. Many types of lesions were treated, warts of all types comprising the largest group. Hæmangiomata are usually frozen with pressure, varying with the depth, until the lesion is white and depressed. A bulla forms at the site. After it is ruptured, an area remains which quickly becomes crusted and which heals rapidly. Leucoplakia, seborrhœa, keratosis and senile keratosis have also been treated. The response of leucoplakial lesions of the mouth and genitals to treatment with liquid oxygen has been satisfactory. Mucosal lesions are dried and frozen thoroughly. Scarring is minimal and of the soft pliable variety. Keloids are not produced by the use of liquid oxygen in persons who have keloids which developed after other forms of treatment.

Vitamin A in Darier's Disease.

Z. A. LEITNER AND T. MOORE (*The British Journal of Dermatology and Syphilis*, February, 1948) describe six cases of Darier's disease and report the results of treatment in which the oral administration of vitamin *A* was combined with exposure of parts of the affected skin to Grenz rays. The literature on Darier's disease is reviewed. In three cases the initial blood vitamin *A* levels were very low: 7, 20 and 28 international units in 100 millilitres of blood, as compared with an average of 120 international units for normal subjects. In two others the blood vitamin *A* level was rather low, the lowest recorded value being 55 international units in each case and the mean values for all estimations before dosing being 78 and 86 international units respectively. In the sixth case the mean value of 99 international units before treatment was not significantly below the mean for normal subjects. They did not find any constant relation between blood carotene and vitamin *A* levels, nor between blood carotene levels and clinical manifestations. Some impairment of liver function was found in the first five cases. There was a high dark-adaptation threshold in the second case, and this became normal after vitamin *A* medication. The authors conclude that Darier's disease is probably not due to a simple avitaminosis. In assessing the results of vitamin *A* treatment the possibility of spontaneous remissions is stressed. The possibility of associated liver damage is discussed. Grenz-ray treatment improved local lesions temporarily in all patients.

Dermatological Manifestations in Psychiatric Disorders.

T. CORNBLEET AND M. BROWN (*The Journal of the American Medical Association*, January 17, 1948) state that several dermatological symptoms are found only in the presence of mental illness. Itching and paræsthesiæ may be indicators of accompanying mental disorder, though, of course, are most often due to primary dermatological changes. The most frequent sites of these unpleasant sensations are the scalp, face, neck, arms, hands and abdomen. The disturbances and sensations cannot be accounted for by allergic or metabolic studies or traced to any rational source. Excessive washing or skin cleansing may be another expression of psychiatric disorder. Such action, considerably beyond what obvious soiling can justify, should always arouse suspicion. It usually springs from fear of contracting syphilis or other disease from contact with familiar objects, such as door knobs, eating utensils or clothing. These ablutionary pursuits may involve the scalp, the genitalia or the entire body. The presenting symptom to the dermatologist in these cases is a dermatitis of varying grades of acuteness. The most common manifestation is a dermatitis with glove distribution. Simply to remember that such cutaneous changes may overlie a disordered mind often helps to clarify a syndrome which would otherwise be obscure. Excessive concern over what most persons come to accept as of the normal pattern, such as thinning of the hair of middle age or even baldness, the appearance of a few additional facial hairs, nævi, dryness of the skin or its oiliness *et cetera*, characterizes a large number of persons familiar to every dermatologist who seek relief for normal or physiological changes about which they become obsessed. The antithesis to this last group of subjects is the one that is inordinately indifferent to and negligent of dermatological disease of the kind which should call for serious attention. Many patients believe their skin to be infested or the seat of parasites, insects, germs or bizarre inanimate materials. Self-induced injuries should excite interest in the patient's neuropsychiatric condition. A few despoil themselves for sympathy; the majority, however, have no apparent reason or desire to mutilate themselves, nor do they understand the significance of their acts. The authors discuss four common psychiatric disorders associated with dermatological symptoms, namely, schizophrenia, manic-depressive depression, psychoneurosis and personality disorders.

Spindle-Cell Epidermoid Epithelioma Simulating Sarcoma in Chronic Radiodermatitis.

C. F. SIMS AND N. KIRISCH (*Archives of Dermatology and Syphilology*, January, 1948) state that it is well known that epithelioma is a frequent sequel to chronic radiodermatitis: between 5% and 25% according to MacKee and Cipollaro. The neoplasm is usually, if not always, of the squamous-celled or prickle-celled variety. The authors call attention to another type of epidermoid epithelioma that occurs in patients with and which is probably caused by chronic radiodermatitis. The neoplasm is composed of spindle-shaped cells and histologically resembles spindle-celled sarcoma and fibrosarcoma. There have been numerous reports of sarcoma in chronic radiodermatitis. It is possible that most of these reported sarcomata were really spindle-celled epitheliomata. The differentiation is not easy, but with the Van Gieson stain and silver impregnation it is possible to prove that the neoplastic cells are of epidermal rather than of connective tissue origin. The authors report two cases of chronic radiodermatitis of the face of many years' duration. Both tumours closely resembled sarcoma histologically. The spindle cells are derived from the prickle-cell layer of the epidermis, and their appearance has changed, so that they take on an elongated, narrow, fusiform appearance, resembling spindle-shaped connective tissue cells. These morphological changes may be due to the associated sclerosis and fibrosis that are present in the skin affected by X rays, the squeezing and compression effects altering the cell shapes. The occurrence of true sarcomata developing in skin affected by X rays or radium is a rarity.

Cysts, Sinuses and Fistulæ.

N. P. ANDERSON (*The Journal of the American Medical Association*, November 8, 1947) states that there is a group of cysts, sinuses and fistulæ which are of dermatological importance. Epidermal cysts are practically always mistakenly diagnosed as sebaceous cysts or wens. True sebaceous cysts are lined with secreting sebaceous cells and contain a fatty or sebaceous material with granules of fat and crystals of cholesterol. There is a peculiar characteristic rancid cheesy odour emanating from this fatty secretion when such a lesion is opened. In contradistinction, an epidermal cyst generally has little or no odour to its contents unless secondarily infected, and when a fragment is burned it presents an odour like burning hair, wool or feathers. The microscopic picture is that of thick layers of keratin throughout. However, the periphery consists of stratified epithelium. Epidermal cysts are

generally found in the cutis or corium and are only rarely situated below the cutis in the subcutaneous tissue. Sebaceous cysts are often impossible to distinguish from epidermal cysts. Actually they are retention cysts of sebaceous glands and hence their epithelial lining is composed of secreting sebaceous gland cells. The therapy of epidermal and sebaceous cysts is the same and the author mentions several different procedures, such as (i) surgical removal and blunt dissection, (ii) injection of 2% procaine solution beneath the skin of the cyst followed by injection into the fibrous capsule and then incision and expression of the sebaceous material, (iii) incision and injection of tincture of iodine after complete evacuation of the sebaceous material by pressure, (iv) incision and swabbing out with 95% phenol solution. Synovial cysts is the term given to a characteristic pea-sized lesion occurring on the dorso-lateral aspects of the fingers and in association with the distal inter-phalangeal joints. This may be treated with X rays or solid carbon dioxide pencil for thirty to forty seconds with firm pressure. Sweat duct cysts or hydrocystomata occur in the cheeks as solitary, pea-sized, shiny, translucent lesions. These can be treated by electro-desiccation. Traumatic epithelial cysts are generally associated with previous trauma. Mucous cysts often situated in the inner surface of the lower lip opposite the cuspid tooth are very common. They should be treated by thorough destruction with electro-desiccation. Dermoid cysts often occur near the inner or outer canthus of the eye. Suppuration in the dermoid cyst which opens may lead to sinus formation. Sinuses of dental origin are usually associated with an alveolar abscess at the root of a tooth. They may open on the chin, the cheek, the floor of the nose, near the orbit and on the buccal and lingual aspects of the gums. The condition of congenital auricular fistula is important to the dermatologist. In its simplest form there occurs a small pimple usually situated at the anterior border of the ascending limb of the helix.

UROLOGY.

Streptomycin in Urinary Tuberculosis.

THE Veterans Administration of the United States Army and Navy (*The Journal of the American Medical Association*, November 8, 1947) reports on the effects of streptomycin in over nine hundred patients with tuberculosis studied since June, 1946. Before May, 1947, the antibiotic had been used in 41 cases of genito-urinary tuberculosis. All patients had multiple lesions of the tract, and all lesions were bacteriologically proved by culture. Sixty-three of the patients completed one hundred and twenty days' treatment in this period. The majority showed considerable relief of vesical disease and vesical symptoms. The same applied to the posterior urethra, prostate and seminal vesicles, while all open epididymal sinuses healed up. There were, however, very few changes in the retrograde or excretion pyelograms. After the first forty days of treatment, in only four of the thirteen cases was a culture grown. The conclusion was that time alone would show whether the relief of the lower urinary tract and genital lesions would be maintained. The same applies to the sterilization of the urine which has been obtained. The disadvantages of this therapy are: (i) toxicity, the antibiotic having to be discontinued in 6% of all cases on account of eighth cranial nerve damage (vertigo), local irritation, fever, jaundice, blood dyscrasias, sensitivity reactions and minor renal damage; and (ii) development of resistance. The latter is a serious problem, for once developed it may be permanent, so that the patient may remain forever debarred from the benefit of streptomycin therapy; the phenomenon is not well understood at present. The dosage used throughout most of this general series of 900 patients has been 1·8 to 2·0 grammes of streptomycin for one hundred and twenty days.

Vesical Diverticulum.

H. M. SPENCE AND S. S. BAIRD (*The Journal of Urology*, November, 1947) have reviewed the principles underlying the direct management of vesical diverticula. Almost all observers agree that the ætiology must be referred to some form of interference with bladder emptying, resulting in outpouching of the mucosa through areas of congenital weakness in the bladder musculature. Of 35 cases studied, seven were in women and one in an infant. It is apparent in most cases that the diverticulum causes no symptoms until infection supervenes. Occasionally the diverticulum is complicated directly by the presence within itself of calculus or neoplasm. For small, wide-mouthed, freely emptying diverticula, relief of the bladder-neck obstruction usually suffices, but under opposite conditions the diverticulum must be suppressed. It is usually unnecessary to undertake the severe operation of complete diverticulectomy; removal of the diverticular mucosa suffices. The separation of the mucosa from the fibrous wall of the sac is easy and bloodless. It is easier to carry out this manœuvre through an incision in the superior wall of the diverticulum than transvesically by way of the neck of the diverticulum. The finger, or a Hagner bag or other instrument may be passed transvesically to enter the diverticulum and so make it more obvious for surgical manœuvres. As the experience of the authors has increased they have found that they get quicker and better results by attaching the sac before dealing with the bladder-neck obstruction.

Sulphathalidine in Coliform Urinary Infections.

H. S. EVERETT, G. A. VOSBERG AND J. M. DAVID (*The Journal of Urology*, January, 1948) discuss their experiences with phthalylsulphathiazole (called sulphathalidine for short) in urinary tract infections with *Escherichia coli*. This drug has been introduced as a successor to sulphasuxidine (succinyl-sulphathiazole) in intestinal infections and has the great advantage that the large doses necessary with sulphasuxidine can be provided with the new drug. The reduction in dosage is to one-third or even one-fourth. Sulphathalidine rarely causes toxic manifestations and is an effective bacteriostatic agent for coliform organisms and clostridia in the intestinal tract. So far as the urinary tract is concerned, the best dose is one gramme every six hours over a period of three weeks. The drug is not effective against other urinary tract invaders than *Escherichia coli*, but is very effective against this germ, so much so that it often succeeds when other sulphonamides have failed. It is also tolerated well by patients who have reacted badly to other sulphonamides and by patients with anæmia, leucopenia or low renal efficiency. So far as mode of action is concerned, it seems that the tissues of the urinary tract are given an opportunity to rid themselves of the existing infection because the constant source of bowel infection is temporarily eliminated.

A New Method for Control of Urinary Tuberculosis.

G. E. SLOTKIN (*The Journal of Urology*, December, 1947) states that this study was undertaken to determine whether there existed a more rapid and permanent means of control of bilateral renal tuberculosis than present methods offer. With the advent of streptomycin such a control seemed to be available, but it was found that as regards the *Mycobacterium tuberculosis* streptomycin was bacteriostatic rather than bactericidal; the effect is only suppressive, and lasting benefit subsequently depends on the resources of the body itself. Tubercle bacilli have a strong tendency to develop resistance against streptomycin, and this usually occurs about one month after the beginning of treatment. Since the advent of the electronic microscope it is well known that the *Mycobacterium tuberculosis* has a protecting waxy cell wall. The same condition occurs exactly in the *Mycobacterium lepræ*. As early as ten years ago, the author used chaulmoogra oil and its derivative esters in a few cases to try to dissolve the cell envelope. Because of the instability of the preparations used, the results in cases of tuberculosis were distressing and unsatisfactory. The idea persisted, however, up to a little before the advent of streptomycin, when a more stable and refined preparation was discovered by the firm of Burroughs Wellcome. The refined oil, which is painless and readily absorbed, is derived from the ethyl ester of hydnocarpus oil and goes under the trade name of "Moogirol". The present studies have been made since January, 1947. on six subjects of active bilateral renal tuberculosis with secondary bladder symptoms, and were undertaken *in vitro* through guinea-pig inoculations and *in vivo*. The results have been so striking that the authors believe they have attained a rapid method of control of this disease. In a period of thirty days positive results in these proven cases have become negative bacteriologically, culturally and chemically. Each patient received one millilitre of the oil intramuscularly for three days, then two millilitres per day for fourteen days. From the seventh day the patient received one millilitre of the oil and one gramme of streptomycin per day for thirty days. In the administration of the streptomycin, the antibiotic is added to sixteen millilitres of distilled water, and injected in eight doses of two millilitres each every three hours. All patients were kept in hospital, but were ambulatory.

British Medical Association News.

SCIENTIFIC.

A MEETING of the South Australian Branch of the British Medical Association was held on February 26, 1948, at the Verco Lecture Theatre, Institute of Medical and Veterinary Science, Frome Road, Adelaide, DR. F. L. WALL, the President, in the chair.

Jaundice.

DR. M. E. CHINNER read a paper entitled "Some Aspects in the Management of a Case of Jaundice" (see page 69).

DR. A. C. MCEACHERN, in opening the discussion, said that from the surgeon's viewpoint the physician sorted out cases of jaundice into hæmolytic, toxic and infective and obstructive, and he handed the obstructive cases to the surgeon for him to deal with the mechanical defect. In a proportion of cases, in spite of clinical and laboratory investigation, it was difficult to be certain whether the jaundice was obstructive or whether it belonged to the toxic and infective group. In such doubtful cases also the patients tended to be referred to the surgeon for exploratory operation. At the meeting they were specially interested in learning something about the various laboratory tests; but he (Dr. McEachern) felt that their interest must be tempered by the knowledge that there were many pitfalls in interpretation, and that several of the tests were still in the experimental stage. The clinical examination of a jaundiced patient was still the sheet-anchor in diagnosis, and he believed that they must stress that continuously to their students. One important pitfall in laboratory diagnosis was that in the advanced case of obstructive jaundice liver damage was superadded, with the production of a dual state. Similarly in an advanced case of toxic or infective jaundice, obstruction of bile passages in the liver occurred.

Dr. McEachern went on to say that in the differential diagnosis of obstructive jaundice they should remember that absence of pain did not necessarily exclude a stone in the common duct, and that the presence of pain did not exclude carcinomatous obstruction. In nearly half the cases of carcinomatous obstruction, pain of some degree occurred; this had been pointed out by T. M. J. d'Offay in *The British Journal of Surgery*, Volume XXXIV, 1946, at page 116. The surgeon in his pre-operative scrutiny of the case must remember that hydatid disease of the liver might cause jaundice, and that occasionally an amœbic abscess of the liver might do likewise. Jaundice occurring in the course of chronic abdominal sepsis was more likely to be due to toxic hepatitis. It was known to them all that errors in diagnosis in cases of jaundice sometimes occurred, and it was interesting to note that F. W. White, an American worker, reviewing 175 cases of jaundice, found that half the errors were associated with unsuspected carcinoma in some form or other (*International Abstracts of Surgery*, Volume LXXVIII, 1944, page 495).

Discussing pre-operative tests, Dr. McEachern said that the depth of jaundice had a considerable bearing on the risks of operation. The deeper the jaundice, the greater the risk, and this was well illustrated in a graph designed by Sir John Fraser in reviewing a large series of cases. This showed a rapid rise in the mortality rate when the icteric index exceeded 100 (*The British Journal of Surgery*, Volume XXVI, 1938, page 393). Certain surgeons had for many years investigated the blood urea content in these cases. The figure was invariably raised in the later stages of severe jaundice, and rose steeply soon after the relief of the obstruction. Renal function was intimately related to liver function, and the outcome of the surgical treatment of obstructive jaundice was dependent to a large extent on the combined function of liver and kidney.

With regard to the stage at which operation should be advised, Dr. McEachern said that no one was infallible in the differential diagnosis of obstructive jaundice, and a clinical diagnosis of carcinoma was no justification for withholding operation. It might be wrong, and even if the cause of the jaundice was a carcinoma, a palliative short circuit operation might lessen the patient's suffering. Early operation was, of course, indicated in every case of obstructive jaundice. Jaundice due to stone in the common duct was a subacute emergency, and operation should be carried out before the supervention of liver damage or infection. On the other hand, operation should not be considered so long as it was thought that the condition was of toxic or infective origin. However, if jaundice of uncertain nature had persisted unabated and unexplained for six weeks, the possibility of an obstructive cause must again be considered and exploration advised without further delay.

Dr. McEachern then said that in preparing the jaundiced patient for operation some of those present no doubt had had the experience of finding a prolonged blood coagulation time which had failed to respond to the administration of vitamin *K* (J. G. Allen, *International Abstracts of Surgery*, Volume LXXVI, 1943, page 401). Dr. Chinner had already told them the reason for this, and it was in those cases especially that blood transfusion was needed. Absence of bile from the intestine interfered not only with the absorption of vitamin *K*, but also with that of the other fat-soluble vitamins, *A* and *D*. Partial night blindness could be demonstrated in many cases of obstructive jaundice; it was due to lack of vitamin *A*. Dr. Chinner had told them that a raised serum phosphatase level was commonly found in obstructive jaundice, and it was probable that this was due to lack of vitamin *D* (M. F. A. Woodruff and R. D. Wright, *The Australian and New Zealand Journal of Surgery*, Volume X, 1940, page 135).

Dr. McEachern went on to say that if at operation the liver was found to be nodular and there was no evidence of obstruction, then the jaundice was toxic or infective in origin, and the surgeon retired after performing a biopsy of the liver. Dr. Chinner had mentioned the risks of a needle biopsy of the liver. Biopsy at operation, though safer, was not entirely devoid of risk, and Dr. McEachern knew of at least one case in which death occurred from hæmorrhage. It was suggested that a biopsy made longitudinally in the free sharp margin of the liver was safer than a V-shaped or wedge excision. If the jaundice was found to be due to obstruction, the cause was removed if possible; or if it was irremovable, a short circuit between the bile passages and the alimentary tract would probably be carried out. Just as the relief of chronic retention of urine might precipitate uræmia, so a sudden decompression of the obstructed bile ducts was fraught with the risk of liver failure, and this was the most important single factor in the high mortality rate affecting that group of patients—even more important than the risk of hæmorrhage. In Sir John Fraser's review of 1035 cases of jaundice due to pancreatic disease, the operative mortality rate was 40%. Dr. W. A. Hailes (*The Australian and New Zealand Journal of Surgery*, Volume XI, 1941, page 24) had stated that in some clinics operation for stone in the common bile duct in the presence of white bile carried a mortality rate of 30%. The lesson to be derived from these figures was that operation should be undertaken early in obstructive jaundice, and that in operating on all jaundiced patients they must take into account the risk of sudden decompression and prepare the patient in every possible way beforehand. When a patient succumbed after the relief of biliary obstruction, death might occur at about forty-eight hours with hyperpyrexia, vomiting, hæmatemesis and coma. This was the so-called "liver death". If the patient survived this crisis, toxic products from the damaged liver cells might affect the renal epithelium and cause fatal anuria about the fifth day. This was well shown in a graph given by Sir John Fraser in the article previously cited; the graph covered the time incidence of all causes of death in 135 fatal cases.

In conclusion, Dr. McEachern said that Dr. Chinner and he had in effect traced some aspects of the story of a patient suffering from jaundice as he passed through a general hospital. It had been said that the surgeon opening a discussion on jaundice had one duty and one duty only—namely, to urge early operation in obstructive jaundice. If they added to this all the newer diagnostic aids and more meticulous preparation of the patient, he thought that they could confidently expect a better prognosis in this very serious clinical condition.

DR. K. STUART HETZEL complimented Dr. Chinner on his paper and Dr. McEachern on his remarks on what was too often a difficult problem—the elucidation of a case of jaundice. Dr. Hetzel stressed the still paramount importance of a meticulous clinical history and examination, and then the importance of obtaining help from special tests—not from any single test, but rather from a number of tests, in such a case as Dr. Chinner had indicated. However, even then there was a moderately high percentage of cases in which the diagnosis could not be made with any certainty. The problem was the distinction between obstructive jaundice and hepatitis with some of the features of obstruction. No one cared to operate on patients suffering from hepatitis, because they stood anæsthesia and operation badly. The difficulties were exemplified by the case of a woman, aged forty-two years, with intense jaundice, who was still in the Royal Adelaide Hospital. Her history did not give much support to the presence of an obstruction of the common bile duct by stone, although this was considered the most likely

cause. The tests carried out gave the following results: the cephalin, cholesterol and flocculation tests produced negative results; the response to the galactose tolerance test and the level of proteins in the serum were normal; the test for urobilinogen produced a negative result, and the amount of alkaline phosphatase in the serum was 35 King-Armstrong units. The plasma prothrombin concentration was 5%; and after the administration of 5·0 milligrammes of vitamin *K* in twenty-four hours it was over 50%, rising to 100% in the next few days. The urine contained gross amounts of bile and the fæces were light in colour. At operation no obstruction was found, and the gall-bladder was flaccid, as were the main bile ducts. Examination of a section of liver removed at operation revealed no hepatitis, and the appearance was consistent with a diagnosis of obstructive jaundice. Dr. Hetzel said that while nowadays one heard little of catarrhal jaundice as originally described by Virchow, probably a few cases of this condition still occurred. Not all of them were cases of infective hepatitis. The case under discussion might be a case of catarrhal jaundice in the old sense, with a catarrhal obstruction of the main bile duct. He could not tell how to save from operation such a patient, who was growing more deeply jaundiced. Finally, many had stressed the dangers of exploratory puncture of the liver to obtain material for histological examination—dangers which were real in inexperienced hands; but he (Dr. Hetzel) wished to bring to the notice of the meeting the relative safety of taking out a small piece of liver in the way Dr. McEachern had indicated under local anæsthesia. Hæmostasis could be obtained, and naturally vitamin *K* had to be given to bring the prothrombin time back to normal before the section was taken. Dr. Hetzel said that he had seen this method successfully used by his surgical colleagues in China.

DR. M. W. ELLIOTT asked Dr. Chinner his opinion of the icteric index in cases of infectious hepatitis as seen amongst servicemen as a guide to the need for absolute bed rest for such patients. Dr. Elliott said that the question arose from his experiences with the handling of some numbers of such patients and from his attendance at small medical conferences in an American general hospital while he was in Japan. The Americans held that while the icteric index was above normal the patient must be confined to bed, despite his sense of well-being; this period of bed rest sometimes lasted for six months. On the other hand, Dr. Elliott said that he had in a convalescent depot dealt with many patients who had icteric indices above normal. They exercised, played games, had seven bottles of beer a week and took a normal, full diet. These patients did well without exception, and were discharged from the convalescent depot as "class A1" after three weeks' intensive physical work.

Dr. Wall, from the chair, thanked Dr. Chinner for his paper and those who had taken part in the discussion for their remarks.

Post-Graduate Work.

THE MELBOURNE PERMANENT POST-GRADUATE COMMITTEE.

PROGRAMME FOR AUGUST, 1948.

THE Melbourne Permanent Post-Graduate Committee announces the following programme for August, 1948.

Week-End Course at Mildura.

On August 7 and 8 the following demonstrations will be conducted at Mildura:

Saturday, August 7: 2 p.m., "Minor Injuries of the Hand", Dr. C. A. M. Renou; 4 p.m., "Diagnosis and Management of the Diarrhœas", Dr. R. Andrew.

Sunday, August 8: 10 a.m., "Rehabilitation of Midwifery Patients", Dr. D. F. Lawson; 2 p.m., "Problems in Pædiatrics", Dr. H. Williams.

The fee for the course is £2 2s., and Dr. Robey, of Red Cliffs, Victoria, will make enrolments. Members of surrounding subdivisions of the Victorian Branch of the British Medical Association are also invited to attend.

Refresher Course in Gynæcology and Obstetrics.

A refresher course in gynæcology and obstetrics will be conducted from August 16 to 28, at the Women's Hospital, Carlton. This will consist of daily ward rounds conducted in groups, during which the routine work of the hospital will be demonstrated and taught. In addition there will be the following lecture-demonstrations:

Monday, August 16, at 9.30 a.m., "Pathology", Dr. H. F. Bettinger; Tuesday, August 17, at 2 p.m., "The Newborn Baby", Dr. Kate Campbell; Wednesday, August 18, at 9.30 a.m., "Genital Prolapse", Dr. W. I. Hayes; Thursday, August 19, at 9.30 a.m., "Puerperal Sepsis", Dr. A. M. Hill; Friday, August 20, at 9.30 a.m., "Disproportion: Trial Labour", Dr. W. Lemmon; Monday, August 23, at 9.30 a.m., "Pathology", Dr. H. F. Bettinger; Tuesday, August 24, at 9.30 a.m., "Toxæmias", Dr. R. Rome; Wednesday, August 25, at 9.30 a.m., "Operative Obstetrics", Dr. John Green; Thursday, August 26, at 2 p.m., "Sterility", Professor J. W. Johnstone; Friday, August 27, at 9.30 a.m., "Ante-Partum Hæmorrhage and Post-Partum Hæmorrhage", Dr. D. F. Lawson.

The fee for the course is £10 10s., and residence at the hospital, which is advisable, is £5. It will be necessary for those attending to demonstrate absence of hæmolytic streptococcal organisms from a throat swab before commencing the course. Arrangements for this procedure are made at the Women's Hospital through the Melbourne Permanent Post-Graduate Committee.

General Refresher Course.

A general intensive refresher course will be held from September 6 to 17. This will coincide with lectures by Professor J. C. Spence, Nuffield Professor of Child Health, of Newcastle, England. Details of these two courses will be published shortly.

Course in Renal Disorders.

A course in renal disorders, suitable for the M.D. Part II and M.R.A.C.P., which commenced in July, will be continued.

Course for D.G.O. Part II.

The course for the D.G.O. Part II—lectures in gynæcology and obstetrics, pathology and bacteriology—which commenced in July will also be continued.

Radiotherapy and Electrotherapy.

Radiotherapy and electrotherapy lectures will be given on Fridays at 9 a.m. by Dr. R. Kaye Scott. These are suitable for the D.T.R.E. Part II.

Enrolments.

Enrolments for metropolitan courses should be made with the Secretary of the Melbourne Permanent Post-Graduate Committee, 426, Albert Street, East Melbourne. Telephone: JM 1547.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Film Evening.

THE following films will be shown at 8 p.m. on Friday, July 30, 1948, at the Stawell Memorial Hall of the Royal Australasian College of Physicians, 145, Macquarie Street, Sydney: "Patent Ductus Arteriosus", "Total Gastrectomy" by Sweet, "Recreational and Occupational Therapy", "Support of Paralysed Face with Fascia". All members of the profession are invited to attend, and further inquiries should be made by communicating with the Secretary of the Post-Graduate Committee in Medicine, 131, Macquarie Street, Sydney. Telephone: B 4606, BW 7483.

Seminar in Medical Statistics.

During Dr. H. O. Lancaster's absence abroad, Miss Helen Newton Turner, of the Section of Mathematics of the Council for Scientific and Industrial Research, will be conducting the monthly seminars in medical statistics. On Wednesday, August 11, 1948, Miss Turner will conduct the first of a series on "Measurements of Association between Two Variables", entitled "Straight Line Relationships", at 5.45 p.m. at the School of Public Health and Tropical Medicine, University Grounds. Any workers in medical or related sciences are welcome to attend.

Courses for Degrees and Diplomas.

Post-graduate courses of ten weeks' duration for Parts I of the examinations for M.S., D.G.O., D.L.O. and D.O. will begin on September 6, 1948. Those intending to make

application to attend these courses are requested to communicate with the Course Secretary, the Post-Graduate Committee in Medicine, 131, Macquarie Street, Sydney, not later than August 18, 1948.

UNIVERSITY OF ADELAIDE POST-GRADUATE COMMITTEE IN MEDICINE.

Overseas Lecturer for 1948.

PROFESSOR J. C. SPENCE, M.D., F.R.C.P., Nuffield Professor of Child Health, University of Durham, Newcastle-on-Tyne, will be in Adelaide from August 2 to 11. During his stay he will deliver three lectures as follows: Wednesday, August 4, "Tuberculosis in Childhood"; Friday, August 6, "Nephritis in Children"; Saturday, August 7, "British Experience of Swift's Disease". These lectures will all be given in the Verco Lecture Theatre at the Institute of Medical and Veterinary Science at 8.30 p.m. Professor Spence has always been associated with pædiatric medicine and teaching. He has been invited to visit Australia by the Post-Graduate Committees of the various States. South Australia's contribution towards his expenses will be £200. Admission to these lectures is by production of an annual ticket, the cost of which is £2 2s., or by payment of a fee of 10s. per lecture at the door. Annual tickets are available from the office of the Post-Graduate Committee, Institute of Medical and Veterinary Science.

Week-End Course in Pædiatrics.

A course will be held on Saturday, August 7, and Sunday, August 8, at the Adelaide Children's Hospital. The programme will be as follows:

Friday, August 6: 8.30 p.m., "Nephritis in Children", Professor J. C. Spence.

Saturday, August 7: 9.30 a.m., "Medical Emergencies of Newborn", Dr. E. B. Sims; 10.15 a.m., "Surgical Emergencies of Newborn", Dr. D. G. McKay; 11.15 a.m., "Common Deformities in Children and their Treatment", Dr. Neville Wilson; 2.30 p.m., "Staphylococcal Infections in Childhood", Dr. T. Grant and Dr. C. O. Rieger; 4 p.m., "Acute Tracheobronchitis", Dr. Ivan Magarey; 8.30 p.m., "British Experience of Swift's Disease", Professor J. C. Spence.

Sunday, August 8: 10 a.m., clinical meeting.

Professor Spence has intimated that he will be in attendance during this course. The fee for this course will be £3 3s., and members intending to be present are asked to forward a cheque payable to the order of the University of Adelaide to the Medical Secretary not later than August 1, 1948. Any member of the profession will be welcome to come and pay his fee at the door, but in order to facilitate catering arrangements, it is requested that all who possibly can should notify the secretary beforehand.

Professor Spence's lectures given during the week-end course are covered by annual tickets, and for holders of these tickets the fee for the week-end course will be £2 2s.

Course Suitable for M.S. Part II Examination.

A series of lectures in surgery and surgical pathology and a demonstration of selected surgical cases will be held for a period of twelve weeks, beginning on Monday, August 2, 1948, provided there are sufficient applicants for the course. These lectures will be given on Monday, Tuesday and Thursday afternoons at 4.30 p.m. in the Post-Graduate Lecture Room, Institute of Medical and Veterinary Science. This course will be similar to the courses previously held. The fee for the course will be £15 15s. Would anyone who wishes to attend this course please notify the Medical Secretary.

Course in Surgical Anatomy.

Dr. J. R. Barbour will give a series of twelve lectures in the Anatomy School at 4.30 p.m. on Friday afternoons, beginning on Friday, September 3, 1948. The list of subjects will be published in the next circular. The fee for this course will be £3 3s. This course is primarily for general practitioners who are interested in surgery, but is also suitable for candidates for higher degrees in surgery.

Demonstration of Post-Mortem Material.

Demonstration of post-mortem material is carried out by Professor Cleland and staff every Wednesday afternoon at the Institute of Medical and Veterinary Science at 3.30 p.m. The fee for attendance at twelve such demonstrations is £3 3s.

Course for D.G.O. Part II Examination.

A course for candidates for D.G.O. Part II examination will be arranged by the Post-Graduate Committee if there are sufficient applicants. It will probably occupy two afternoons a week and will include pathology, bacteriology, gynæcology and obstetrics. If held, this course will run in September, October and November. Anyone interested is asked to communicate with the Medical Secretary as soon as possible.

Correspondence.

SINUSITIS AND SHORT-WAVE THERAPY.

SIR: I am interested to see letters in THE MEDICAL JOURNAL OF AUSTRALIA of June 19 dealing with certain aspects of a paper given by myself on the above subject. Dr. Woolcock does not approve of radiological evidence of sinusitis and states that the criteria for diagnosis are that (i) one should see pus issuing from a sinus ostium or (ii) find evidence in a sinus washing or make a bacteriological examination of the pent-up secretion. According to Dr. Rosa Ford, who recently published a book on paranasal sinusitis, it is fallacious to assume that unless pus can be demonstrated there is no sinusitis; that if irrigation of a sinus returns no pus there is no sinusitis; and most important of all it is a fallacy to state that thickened mucous membrane does not necessarily indicate active infection. Dr. Dan Mackenzie, who wrote a 600-page text-book on diseases of the ear, nose and throat, says that "the ethmoidal cells are undoubtedly more frequently affected . . . than any other sinuses". If this be the case, Dr. Woolcock, according to his own standards, must either observe pus in the ethmoidal ostia or irrigate the cell and pathologically examine the irrigating fluid before he can satisfy himself that ethmoiditis is present, in each case an impossible undertaking. When the antrum or frontal sinus is irrigated, the irrigating fluid passes through the natural sinus ostium, and before it is recovered it must pass over mucous membrane in the nasal cavities and also over the posterior pharyngeal wall. In this way it could be contaminated by infection in these cavities, as also by pus issuing from another sinus, and thus make invalid the washout. I am not alone in emphasizing the important part X ray plays in diagnosis of sinusitis. Dr. Richard E. Dunn, of Sydney, who distinguished himself by having a paper quoted in "The 1947 Year Book of Ear, Nose and Throat", says: "X-ray examination was found most helpful in diagnosis of sinusitis and also in ascertaining which sinuses were involved." Dan Mackenzie states:

> In the investigation of a case of suspected nasal sinus suppuration useful information can be obtained by means of the X rays. Liquid, such as pus, and perhaps polypi in the antrum or frontal sinus, throw a faint shadow, visible on the developed plate as a dullness of these cavities which render their outlines blurred and indistinct as compared with the normal. Skiography of the sinus demands special skill and experience for which reason the rhinologist ought himself acquire the art of reading the skiagram. It was useful in the diagnosis of antrum and frontal sinus suppuration.

In my work I have correlated radiological evidence with the results of proof puncture and irrigation before, during and after treatment, and I have satisfied myself as to the value of X ray as a diagnostic measure and learned the importance of correctly interpreting skiagrams.

Dr. Woolcock asks would I deny the soundness of draining retained infected secretions. My reply is yes, especially where the conservative treatment outlined in my paper has been instituted. There is no occasion to drain away pus where short-wave therapy is skilfully given. The orthodox conception of the value of drainage is that if pus can be washed away from the overburdened mucosa, the sinusal cell will have a chance to regenerate and the cilia an opportunity to function. In practice no sooner is lavage of the sinus over than the patient commences sneezing, a fresh cold has developed and pus is secreted into the sinus as copiously as ever. In reply to the question as to whether I treat appendicitis, cholecystitis and brain abscess with short-wave therapy, I have to state that the first line of treatment for sepsis in any part of the body is the employment of short-wave, provided always that it is personally given by a doctor who understands dosage and spacing between treatments and is a sound clinician. I do not advocate its general use in appendicitis, nor do I think

that a layman should be asked to treat infective conditions with physical therapy. Dr. Woolcock credits me with claiming that once an asthmatic's sinuses are radiologically clear of infection there should be no further attacks of asthma, and he makes other statements unsupported by evidence to which I shall not reply.

I believe my paper sufficiently refutes the statement that I am putting short-wave therapy forward as the answer to all types of sinusitis as a single measure of treatment. That is the last impression that I want to leave.

Actually it has become common for practitioners to send patients to anyone with short-wave apparatus with the instruction: "Give him short-wave." That must account for failures.

Yours, etc.,

WILKIE SMITH.

Sydney,
June 25, 1948.

SIR: In reference to Dr. R. H. Bettington's letter published June 9. Following Dr. Wilkie Smith's technique there are failures to restore normal X-ray appearances after surgical intervention and in very long-standing cases, but there are no failures in symptomatic relief and the improvement of general health. The method includes antibiotic drugs, dietetic, vaccine, anti-allergic, and general measures when indicated, together with properly applied short-wave diathermy. This is hardly "one treatment" in application and there is no "theory" as regards good results. Dr. Bettington's "chronic cases in which acute exacerbations followed short-wave therapy" were unfortunate in being either *post hoc* or, if *propter hoc*, in having improperly applied short-wave therapy. How to obviate trauma to the inflamed cell by electrotherapy is elementary teaching in any school of physical medicine.

Those of us who have been operating on the nasal sinuses for many years, and in some cases have had operations on our own sinuses, have no good reason to feel smug concerning our results. Instead of being harsh critics our attitude should be to learn all we can from one who claims such great improvement in results, particularly when backed by the wealth of clinical material under Dr. Wilkie Smith's care.

Yours, etc.,

JOHN A. SHANASY.

149, Wickham Terrace,
Brisbane,
July 1, 1948.

THE PHARMACEUTICAL BENEFITS ACT.

SIR: As two of the practitioners who availed themselves of the freedom of action left open to members by the British Medical Association Federal Council in deciding to work the Federal Government pharmaceutical benefits scheme, we feel that our experiences of one month of the scheme in action will be of interest to our profession.

We have found that our freedom to prescribe according to the principles of modern therapeutics has been very considerably increased by our use of the scheme for the reason that we no longer have to consider whether or not our patients can afford to buy particular drugs. This factor has in the past acted against patients who could not afford, for example, a full course of sulphonamides or penicillin in oil. We have found, for example, that we can now effectively treat paronychia with three daily injections of 300,000 units of penicillin in oil at no extra cost to the patient, whereas formerly the nail was removed surgically and the patient more or less incapacitated for a fortnight.

The harmful effect of the economic factor must be greater in most centres where the general income level is much below that of Broken Hill. Consequently the benefits to patients of the government scheme would also be greater.

We have found that at least 90% of our prescriptions can be filled from the formulary as it stands, assessing ourselves as having about average ability to apply modern therapeutics. There are defects in the formulary; some preparations are allowed, in our opinion, in too small quantities; a few necessary drugs such as sulphacetamide and the antihistamine drugs have been omitted to date. Taken as a whole, however, the formulary is remarkably comprehensive. It can, of course, be kept up to date and improved, particularly if practitioners send suggestions based on their experiences of its use to the appropriate body.

We have not found the extra writing involved in the official prescriptions to be more than writing the address of the patients (which in any case has been required for prescriptions of dangerous drugs), nor does the production of a duplicate prescription entail any more work than flicking over a simple carbon paper system.

We have found the writing of separate prescriptions, where more than one preparation is required for the one patient, a little irksome, but by no means a major time-waster.

We have, of course, been able to prescribe outside the formulary when necessary because of the non-inclusion of preparations, or more frequently their non-inclusion in combinations we have become particularly attached to, or more frequently still because the patient has become attached to a particular proprietary line. But we stress such outside prescribing, in our experience of almost 1400 prescriptions, does not amount to more than 10%.

As regards the penal regulations, we think it unlikely we shall be involved in these as they are designed to prevent fraud rather than to make things difficult for the practitioner.

We conclude by stating that in our opinion it would be unwise at the present time to allow completely free prescribing. Such a state of affairs would, of course, be ideal, but without complete nationalization of the drug industry it would be very premature. With the drug industry nationalized there would be some control, for example, on the natural tendency for more or less indiscriminate prescribing of any new drug or preparation whose therapeutic value has not yet been proven.

We feel that the formulary, kept up to date and improved by the application of experience in its use, can help considerably to raise the standard of therapeutics in place of the ever-growing tendency to rely on the blotting paper blurbs of the proprietary firms.

Yours, etc.,

ALAN H. FINGER,
H. J. P. MCMEEKIN.

Broken Hill,
New South Wales,
June 30, 1948.

DIAGNOSIS OF GASTRIC DISEASE: SHOULD RADIOLOGY OF THE STOMACH BE ABANDONED?

SIR: I must point out that in order to study by radiology the profile of the cardiac end of the stomach in the supine position, it is necessary that the patient be given as much opaque meal as can be tolerated. If the meal is insufficient to fill the cardiac end with the patient supine, the appearance on the skiagram may closely resemble the filling defect due to a carcinoma. Since my recent article I have seen two patients in whom needless anxiety has been caused through such insufficient filling. I did not stress this point in my recent article, but included it with the more detailed description in course of publication in *The Australian and New Zealand Journal of Surgery*.

Yours, etc.,

V. J. KINSELLA.

235, Macquarie Street,
Sydney,
July 2, 1948.

STANDARDS AT THE UNIVERSITY OF SYDNEY MEDICAL SCHOOL.

SIR: Your editorial appearing in THE MEDICAL JOURNAL OF AUSTRALIA of June 19 is timely and should be of interest to those who have questioned the standards at the University of Sydney Medical School.

How natural for one to stand up for one's Alma Mater! Osler[1] thought so, as the following extracts from one of his addresses show.

> What, for example, is more proper than the pride which we feel in our teachers in the University from which we graduated, in the Hospital in which we were trained. He is a "poor sort" who is free from such feelings which only manifest a proper loyalty.

The backbone of education at Oxford is the jealously guarded collegiate system. Accommodation is limited, and thus selection of students for all faculties is inevitable. No such collegiate system pertains in the large city universities and any comparison, then, with Oxford is out of place.

As far back as 1903 the Canadians apparently were facing similar problems that today beset Sydney and Melbourne, for Osler[2] offered the following suggestions.

> An affiliation should be sought with every other hospital in the city and provinces of fifty beds or over, in

> each of which two or three extra-mural teachers could be recognized who would receive, for three or more months, a number of students proportionate to the beds in hospital. There are difficulties in the way—but is there anything worth struggling for in this life which does not bristle with them.

In the light of the above remarks the relative number of medical students to beds available for clinical instruction at Edinburgh, Sydney and Melbourne Universities is quoted, particularly as the University of Sydney Medical School is an Edinburgh foundation. Surely no one would be so bold as to criticize the quality of Edinburgh teaching or the products of its university. There is also a vast pool of out-patients available, for it is here that a student's clinical groundwork is laid. In Edinburgh with 1624 medical students there are 2532 beds of public wards of metropolitan hospitals with fifty beds or more exclusive of those for intermediate or private patients;[3] this excludes 779 beds for subjects of infectious diseases and 800 beds for mental patients. In Sydney with 1876 medical students[4] there are 4774 public ward beds of corresponding category,[5] exclusive of those for mental patients and for patients in military establishments. In Melbourne with 1021 medical students[6] there are 3205 such public ward beds, exclusive of 367 beds for convalescents and those for mental patients. The number of new patients attending out-patient departments of metropolitan hospitals with fifty public beds or over during 1946-1947 totalled 553,469 in Sydney[5] and 267,524 in Melbourne.[7] The total attendances at out-patient departments of metropolitan hospitals with fifty public beds or over during 1946-1947 were 1,794,956 in Sydney[5] and 1,036,504 in Melbourne.[7]

It would seem from the figures quoted above, and based on Edinburgh University, that the medical school at the University of Melbourne could expand without loss of prestige or of standards of education and the risk of turning out half-baked doctors. We, who are actively engaged in medical education in Sydney, feel, with the absence of restrictions on the natural flow to and from the university, that the university has grown in moral stature, enhanced its position in leadership and, by careful selection of the teaching staff, raised the standards.

And finally, if young Australians are to be thwarted in their desires for higher or specialized education, Julian Huxley's[8] observations are worthy of notice:

> It is a big job to find ways in which self-assertive impulses can issue along conscious and constructive channels and prevent the accumulation of this store of psychological dynamite.

Yours, etc.,

"Rolna",
Macquarie Street,
Sydney.
June 24, 1948.

HOWARD BULLOCK.

References.

(1) William Osler: An address to the Canadian Medical Association at Montreal, 1902.
(2) William Osler: An address delivered at the University of Toronto, 1903.
(3) "Calendar of the University of Edinburgh", 1946.
(4) The Registrar of the University of Sydney: Personal communication, June, 1948.
(5) "Report of the Hospitals Commission of New South Wales", Second Edition, November, 1946.
(6) The Registrar of the University of Melbourne: Personal communication, April, 1948.
(7) "Twenty-third Report of the Charities Board of Victoria: Annual Report for the Year Ended 30th June, 1947."
(8) Julian Huxley: "Selected Essays: Man in the Modern World", 1947.

SIR: The discussion on the above seems limited to the effect of increased numbers of students on teaching, and satisfaction is expressed on the standards preceding this. Similar satisfaction seems to me to have been expressed since my qualification in 1926, and at least then, in my humble opinion, with inadequate justification. I am a bit out of touch with general training now, but at least know something of the orthopædic standards of the new graduate: in most cases it certainly is "half-baked", has been for years, and what is more, the men themselves tell me they have had little or no undergraduate instruction in orthopædics (including fractures).

Of course, the reply is that specialists lack a sense of proportion, but the existence of a large field for the student to cover has not passed unnoticed, nor have resident appointments in five large hospitals and seven years of general practice before specializing rendered me less fit to make the above remarks. Further, before anything is said about a solitary grumbler, may I quote from the editorial of the last issue of THE MEDICAL JOURNAL OF AUSTRALIA (June 19) on "A British Medical Association Report on Medical Education":

> The recommendations generally speaking reflect the statement by the committee that reform of the medical curriculum is not only a compelling and urgent need, but that the required changes are more radical than is generally conceded,

from which it may appear that my specialty is not alone in exciting anxiety.

May I finish with two constructive suggestions, and though some there are who may laugh at the first, I make it very seriously.

1. There should be study and control of the teachers as well as the taught. I suggest the issue of an annual *questionnaire* to students regarding the efficiency of their teachers and of the course. Any outstanding condemnation should be the subject of inquiry either by a special body appointed for the purpose or by the Senate itself.

2. Training should be carried into hospital resident life where honorary medical officers in all hospitals should be appointed not only to care for patients, but specifically to teach resident medical officers (this would apply more particularly to non-teaching hospitals). I especially recall when a resident at the Children's Hospital that there was a set number of "T's and A's" which each resident was entitled to perform under supervision, not as a favour, but as a right. I suggest that a specified amount of practical surgery under supervision should be laid down as a course for all resident medical officers in their first twelve months. This may, of course, be unnecessary for some hospitals now, where the honoraries look after their residents, but may not be general. In any case, there is another difficulty to overcome, namely, the reluctance of hospital boards to pay for more than a bare minimum of resident medical officers, and we may have to bring pressure to bear, so that sufficient numbers are appointed to allow adequate time for study of both theory and practice.

Yours, etc.,

"Harley",
143, Macquarie Street,
Sydney.
June 24, 1948.

C. C. MCKELLAR.

SIR: Since there has been a certain amount of discussion about the standards at the University of Sydney Medical School it seems appropriate that some comment should come from one of those who are actually concerned with the attempt to educate the students in question.

In the first place, I wish to congratulate Sir Alan Newton on his insistence that "the standards . . . are more than a domestic matter of importance only to the people of N.S.W.". I am convinced that he would have written more strongly still if he were vividly acquainted with the facts in question.

Since my object is to offer evidence at first hand I naturally confine myself to the subject of physiology. Just prior to the war there were 164 students in Medicine II and 98 other students taking Physiology I, making a total of 262. All the work in practical physiology was carried out in two classrooms, one equipped for 60 students, the other for 30 students. This year instead of 262 students we have a total of 796, including 432 medical students. Apart from equipping small galleries in the two rooms mentioned, no additional provision has been made to cater for these students. To give practical work at all, class times have to be cut down and the individual classes hopelessly overcrowded.

It is universally acknowledged that it is essential to provide teachers who keep alive the spirit of education by engaging in some research. Yet we cannot give the teaching Fellows, who are engaged in taking these practical classes, the space they require to carry on research. Four teaching Fellows have between them one room about sixteen feet by nine feet equipped with one table, a few lockers and a few chairs. Eighteen months of reiterated applications are about to result in some bookcases for these teachers.

When I was appointed to take charge of the department of physiology nearly two years ago, I stated the case for the urgent need of more rooms. One room sixty feet by twenty feet was virtually promised by the then Vice-Chancellor. After eighteen months' persisting with this application for such a room which would cost about £2000, the request was finally rejected about one month ago by the university.

I need hardly emphasize the importance of the subject of physiology in the pre-clinical training of the medical student. Until matters here are very greatly improved, I consider

that our students (in comparison with their fellows of the sister universities) will suffer a deplorable hiatus in their fundamental training. This deplorable hiatus cannot be remedied by subsequent teaching, whatever the herculean efforts of their very able educators of the concluding years.

We are suffering from overcrowding and also from neglect. The timely criticism of eminent persons, such as Dr. Poate, Sir Alan Newton and others, should help to keep the stark truth clearly before the public, the governing bodies and the university authorities, and should help to dispel that fatal attitude of satisfaction with a situation which is anything but satisfactory.

Yours, etc.,

F. S. COTTON,
Professor of Physiology.

Medical School,
University of Sydney,
July 1, 1948.

A NOTE ON THE ROYAL ARMY MEDICAL CORPS.

SIR: I have read with interest "A Note on the Royal Army Medical Corps" by my friend Colonel McIntosh in the June 12, 1948, number of THE MEDICAL JOURNAL OF AUSTRALIA. He will, I am sure, not take umbrage if I point out that the name of Wellington's senior medical officer should be spelt McGrigor, not McGregor. Sir James McGrigor was the eldest of three sons of Colquhoun McGrigor, a merchant of Aberdeen. He studied medicine at Aberdeen and Edinburgh. Desiring to become an army surgeon, he went to London, and after further study, obtained by purchase the post of surgeon to the famous Connaught Rangers. His appointment was dated September 13, 1793, and his name was at first spelt in the army list "MacGregor". This explains the spelling of the name adopted by Colonel McIntosh. I possess a fine engraved portrait of Sir James McGrigor, Bart., F.R.S., which I purchased in London at the end of the first world war. Further particulars of Sir James's distinguished career are available in the Dictionary of National Biography.

Yours, etc.,

H. S. NEWLAND.

163, North Terrace,
Adelaide,
July 1, 1948.

Congress Notes.

AUSTRALASIAN MEDICAL CONGRESS (BRITISH MEDICAL ASSOCIATION).

THE Executive Committee of the sixth session of the Australasian Medical Congress (British Medical Association) to be held at Perth from August 15 to 21, 1948, has forwarded the following information.

Additional Baggage Allowance by Air.

To assist members of Congress wishing to bring dress clothes and academic robes, Trans-Australia Airlines has agreed to increase the free baggage allowance to 45 pounds for members of Congress travelling to and from interstate ports. The increased allowance will be made available only to members of Congress and will not apply to any members of their families who may be travelling with them.

Australian Medical Board Proceedings.

TASMANIA.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Act*, 1918, of Tasmania, as duly qualified medical practitioners:

Colin, Ernest, L.R.C.S. and P., 1938 (Edinburgh), L.R.F.P. and S., 1938 (Glasgow), Launceston.

Stokes, Wilfred James, M.R.C.S. (England), L.R.C.P. (London), 1934, M.B., B.S., 1936 (Univ. London), M.D., 1938 (Univ. London), D.R.C.O.G., 1938 (London), M.R.C.P., 1939 (London), Launceston.

Inglis, William, M.B., B.S., 1943 (Univ. Sydney), Campbell Town.

Fisher, Harry Medcalf, M.B., B.S., 1922 (Univ. Adelaide), F.R.C.S., 1930 (Edinburgh), D.G.O. (Trinity College, Dublin), L.M., 1930 (Rotunda), M.R.C.O.G., 1935 (London), F.R.C.O.G., 1947 (London), Launceston.

Howqua, June Louise, M.B., B.S., 1944 (Univ. Melbourne), M.D., 1947 (Univ. Melbourne), Hobart.

Laver, Jack Charles, M.B., B.S., 1934 (Univ. Melbourne), Royal Hobart Hospital, Hobart.

NEW SOUTH WALES.

THE undermentioned have been registered, pursuant to the *Medical Practitioners Act*, 1938-1939, of New South Wales, as duly qualified medical practitioners:

Aitken, William, M.B., B.S., 1939 (Univ. Melbourne), 4, Deakin Parade, Mildura, Victoria.

Henley, Maire, M.B., B.Ch., 1941 (N.U.Ireland), 7, Hampton Street, Croydon Park.

Seifert, Morris, M.R.C.S. (England), L.R.C.P. (London), 1943, 18, Penkivil Street, Bondi.

The undermentioned additional qualifications have been registered:

Halliday, Francis Bathurst, 185, Macquarie Street, Sydney (M.B., B.S., 1940, Univ. Sydney), D.O.M.S., 1947.

Kiely, Roger John, 3, Crown Street, Gladesville (M.B., 1941, Univ. Sydney), D.P.M., 1948 (Univ. Sydney).

Rubinstein, Kusiel, Queen Victoria Sanatorium, Thirlmere (M.D., Paris, 1936; registered in accordance with the provisions of Section 17A of the *Medical Practitioners Act*, 1938-1939), M.B., B.S., 1947 (Univ. Sydney).

Russell, John Donald, 6, Suttie Road, Edgecliff (M.B., Ch.M., 1926, Univ. Sydney), D.P.M., 1947 (Univ. Sydney).

Williams, Dudley Clarence, "Borambel", Redall Street, Manly (M.B., 1939, Univ. Sydney), B.S., 1947 (Univ. Sydney).

Windeyer, John Spencer, 7, Water Street, Wahroonga (M.B., B.S., 1941, Univ. Sydney), Dip. Anæsthesia, 1947 (Univ. Sydney).

Lowe, Gordon Bradley, 141, Macquarie Street, Sydney (M.B., Ch.M., 1915, Univ. Sydney, F.R.C.S., Edin., 1921), M.R.C.O.G., 1935, F.R.A.C.S., 1938, F.R.C.O.G., 1947.

Raymond, Roland Lionel, 223, Miller Street, North Sydney (M.B., Ch.M., 1923, Univ. Sydney, F.R.C.S., Edin., 1926), D.O.M.S., London, 1939.

QUEENSLAND.

THE undermentioned have been registered, pursuant to the provisions of *The Medical Acts, 1939 to 1946*, of Queensland, as duly qualified medical practitioners:

Reid, George Merson, M.B., Ch.B., 1942 (Univ. Aberdeen), D.P.H., 1947 (Univ. Aberdeen), 14, Florence Street, Ascot, Brisbane.

Hill, William Howard, M.B., B.Ch., B.A.O., 1932 (Queen's University, Belfast), c.o. Dr. W. W. Stevens, Charters Towers.

The following additional qualifications have been registered:

Booth, Frederick John, 113, Wickham Terrace, Brisbane, B17, M.R.C.P. (Edinburgh), 1948.

Lynch, John Aloysius, Wickham House, Wickham Terrace, Brisbane, M.S., 1942 (Univ. Queensland), F.R.A.C.S., 1948.

Obituary.

VINCENT WELLESLEY SAVAGE.

THE following appreciation of the late Dr. Vincent Wellesley Savage has been received from Dr. A. H. Moseley.

V. W. Savage and I served together in the 5th Australian Field Ambulance from its formation in March, 1915, until we left Anzac in December, 1915, and I knew of his work as an R.M.O. and ambulance officer in France.

He was a first-rate R.M.O., very cool under fire or in an emergency, and as his professional skill was very great he had the complete confidence of his battalion commander and the real affection of the men.

He was very witty in a dry way and could guild the pill of "M and D" in such a way that the men accepted his verdict cheerfully.

His first job at Anzac was to take stretcher-bearers to clear the wounded after an attack, on the left, by the 18th Battalion. I was with Colonel Roth, our C.O., when he reproved him for taking unnecessary risks, and "Sav's" reply was characteristic: "We were all new to the game so I thought I had better give them a lead out into 'No Man's Land' and then no one could squib it." This was typical of his attitude to his job and he was always where he could do most good for the casualties.

He was a very loyal comrade and an excellent companion in the mess and at work. He was most reliable and we knew that if "Sav" were detailed for a job of work it would be carried out well and without fuss.

HENRY MITCHELL BENSON.

WE regret to announce the death of Dr. Henry Mitchell Benson, which occurred on June 30, 1948, at South Perth.

WILLIAM BROOKES CLIPSHAM.

WE regret to announce the death of Dr. William Brookes Clipsham, which occurred on July 1, 1948, at Grafton, New South Wales.

EDWARD LLOYD PARRY.

WE regret to announce the death of Dr. Edward Lloyd Parry, which occurred on July 3, 1948, at Melbourne.

Nominations and Elections.

The undermentioned have been elected as members of the South Australian Branch of the British Medical Association:

Caust, David Kennings, M.B., B.S., 1946 (Univ. Adelaide), 115, Watson Avenue, Toorak Gardens.

Kenihan, Robert Austin, M.B., B.S., 1946 (Univ. Adelaide), 200, Goodwood Road, Goodwood.

Books Received.

"Hallmarks of Mankind", by Frederic Wood Jones, D.Sc., M.B., B.S., F.R.S., F.R.C.S.; 1948. London: Baillière, Tindall and Cox. 8½" x 5½", pp. 96, with many illustrations. Price: 10s. 6d.

"Recent Advances in Surgery", by Harold C. Edwards, C.B.E., M.S., F.R.C.S.; Third Edition; 1948. London: J. and A. Churchill, Limited. 8" x 5¼", pp. 448, with many illustrations. Price: 24s.

"The Mothercraft Manual or the Expectant and Nursing Mother and Baby's First Two Years", by Mabel Liddiard, Introduction by Dorothy M. Taylor, M.D., D.P.H.; Eleventh Edition; 1948. London: J. and A. Churchill, Limited. 7¼" x 5", pp. 190, with many illustrations. Price: 5s.

"Diseases Affecting the Vulva", by Elizabeth Hunt, B.A., M.D., Ch.B. (Liverpool); Third Edition; 1948. London: Henry Kimpton. 9½" x 6", pp. 212, with many illustrations, some of them coloured. Price: 25s.

"Ophthalmic Nursing", by Maurice H. Whiting, O.B.E., M.A., M.B., B.Ch. (Cantab.), F.R.C.S.; with Introduction by Sir John Parsons, C.B.E., D.Sc., F.R.C.S., F.R.S.; Fifth Edition; 1948. London: J. and A. Churchill, Limited. 7¼" x 4¾", pp. 148, with illustrations. Price: 7s. 6d.

"Handbook of Communicable Diseases for the Use of Medical Officers of Schools (Formerly a Code of Rules)", issued by The Medical Officers of Schools Association; Eleventh Edition; 1948. London: J. and A. Churchill, Limited. 8¾" x 5½", pp. 72 Price: 5s.

"Rheology in Relation to Pharmacy and Medicine", by G. W Scott Blair, M.A., D.Sc., F.R.I.C., F.Inst.P.; 1948. London: The Pharmaceutical Press. 8½" x 5½", pp. 20. Price: 2s.

"Fatty Liver Disease in Infants in the British West Indies", by J. C. Waterlow; 1948. Medical Research Council of the Privy Council, Special Report Series Number 263. London: His Majesty's Stationery Office. 9½" x 6", pp. 96, with illustrations. Price: 2s.

Diary for the Month.

JULY 19.—Victorian Branch, B.M.A.: Finance, House and Library Subcommittee.
JULY 20.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JULY 21.—Western Australian Branch, B.M.A.: General Meeting.
JULY 22.—New South Wales Branch, B.M.A.: Clinical Meeting.
JULY 22.—Victorian Branch, B.M.A.: Executive Meeting.
JULY 23.—Queensland Branch, B.M.A.: Council Meeting.
JULY 27.—New South Wales Branch, B.M.A.: Ethics Committee.
JULY 28.—Victorian Branch, B.M.A.: Council Meeting.
JULY 29.—New South Wales Branch, B.M.A.: Branch Meeting.
AUG. 3.—New South Wales Branch, B.M.A.: Organization and Science Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute; Brisbane City Council (Medical Officer of Health). Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

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